e-ISSN: 0975-1556, p-ISSN:2820-2643

Available online on www.ijpcr.com

International Journal of Pharmaceutical and Clinical Research 2024; 16(6); 1950-1952

Original Research Article

Evaluation of Histopathological Changes in Placenta of Intrauterine Growth Restriction

Homa Imam[1], Hena Jabin[2], Anupama Sinha[3]

[1,2]Senior Resident, Department of Obstetrics and Gynaecology, Jawaharlal Nehru Medical College and Hospital, Bhagalpur, Bihar

[3]Professor and Head of Department, Department of Obstetrics and Gynaecology, Jawaharlal Nehru Medical College and Hospital, Bhagalpur, Bihar

Received: 25-01-2024 / Revised: 23-02-2024 / Accepted: 26-03-2024

Corresponding Author: Dr. Hena Jabin

Conflict of interest: Nil

Abstract:

Background: A term baby cannot be born without a healthy placenta. IUGR is one condition associated with placental insufficiency. IUGR and placental qualitative changes are tightly associated. The aim of the present investigation was to evaluate the histological changes in placentas from IUGR patients.

Methods: The present study was conducted in the Department of Obstetrics and Gynaecology, JLNMCH, Bhagalpur, Bihar from November 2022 to October 2023. A total of 400 patients were studied. Out of them 200 were normal patients without IUGR and 200 with IUGR. These patients were evaluated with the help of semi structured proforma consisting of various socio-demographic and clinical variables.

Results: Number of LSCS deliveries in IUGR patients (59) is significantly more than control group (33). The number of syncytial knot formation (>10) in IUGR is 169 compared to 153 of control and hence is statistically insignificant. Cytotrophoblastic proliferation (>10) in IUGR (126) is significantly more than (92) in the control group. Number Of area fibrinoid necrosis in villi > 5 in IUGR group is 57 compared to control group (29) (significantly higher) No. of hyalinised areas (> 5) in IUGR (101) is significantly higher than in control group (68).

Conclusion: Histopathological observations in low power fields, such as the formation of syncytial knots, Cytotrophoblastic cellular proliferation, calcification, and hyalinization of villi, are similarly identified as typical aging alterations in the placenta; however, they are more common and occur earlier in the IUGR group. In conclusion, the etiological basis for intrauterine growth restriction is these placental morphological and histological findings.

Keyword: Intrauterine growth retardation, Foetus infarction, Fibrinoid necrosis, Placenta, Thrombosis.

Introduction

A fetus that is not able to reach its full growth potential due to endogenous pregnancy-related environmental factors and genetic makeup is said to have intrauterine growth retardation (IUGR).

The mother's delivery of nutrients and oxygen into the umbilical circulation through the placenta is essential for the growth and viability of the fetus. The two main causes of intrauterine growth restriction (IUGR) and fetal mortality are placental factors and hypoxemia.

One disorder linked to placental insufficiency is IUGR. [1] Prematurity and hypoplasia were the primary pathogenic findings in the placenta, and a strong correlation was observed between IUGR and placental qualitative alterations. [2]. The weight of the placenta in IUGR pregnancies was shown to be lower than that of normal placentas in a histological evaluation. In the placenta of IUGR, infarction and intervillous fibrinoid deposits were more prevalent. Furthermore, IUGR placentas were more likely to have cytotrophoblast hyperplasia and basal membrane thickening. The primary histopathological results indicated both fetal blood flow constriction and a decrease in placental blood flow. [3] Knowledge about placental anomalies can help distinguish between acute (peripartum) pressures and chronic fetal injuries. [4]

Materials and Methods

The present prospective study was conducted in the Department of Obstetrics and Gynaecology Jawaharlal Nehru Medical College and Hospital, Bhagalpur, Bihar from November 2022 to October 2023. A total of 400 patients were studied. Out of them 200 were normal patients without IUGR and

200 with IUGR. These patients were evaluated with the help of semi structured proforma consisting of various socio-demographic and clinical variables. All patients in the study were hospitalised. IUGR was taken as baby weight less than 10th percentile of their gestational age. Preterm deliveries were not taken for study. Placenta with cord and membranes were collected immediately after delivery. Any abnormality of cord and membranes was noted. The placentae along with the umbilical cord were preserved in 10% formalin solution (in water).

Each placenta was collected soon after delivery from the labour room or from the operation theatre. Placenta as a whole was kept in formalin solution in a large container with snugly fitting lid. Then specimen was transported to research laboratory of Anatomy department.

Whole of the specimen of placenta was taken out from the large container and kept on a clean flat surface on the dissection table wrapped with a sheet of polythene. The specimen was washed well with normal saline and the membrane was trimmed off with a pair of sharp scissors near the margin. The specimen was soaked with blotting paper and excess blood clot was removed from the surface. The umbilical cord was cut at a distance of 2 cm from its attachment with placenta (Ameren and Dunhill, 1966).

Patients with baby birth weight of < 10th percentile of gestational age, IUGR due to all causes, any age, informed consent were included in this study.

Patients with preterm, lack of consent, damaged placenta were excluded in this study.

Results

Number of LSCS deliveries in IUGR patients (59) is significantly more than control group (33). The number of syncytial knot formation (>10) in IUGR is 169 compared to 153 of control and hence is statistically insignificant. Cytotrophoblastic proliferation (>10) in IUGR (126) is significantly more than (92) in the control group.

Number of area fibrinoid necrosis in villi > 5 in IUGR group is 57 compared to control group (29) (significantly higher).

No. of hylinized areas (> 5) in IUGR (101) is significantly higher than in control group (68).

Table 1: Mode of delivery

Outcome	Control		IUGR	
	No. of cases	Percentage	No. of cases	Percentage
LSCS	33	16.5%	59	29.5%
Vaginal	167	83.5%	141	70.5%
Total	200	100%	200	100%

(Chi square value 11.33, p value 0.0008)

Table 2: Number of syncytial knot formation

No. of Syncytial knot	Control		IUGR	
	No. of cases	Percentage	No. of cases	Percentage
<10	42	21%	31	15.5%
10-25	152	76%	158	79%
>25	01	3%	11	5.5%
Total	200	100%	200	100%

Chi square value 9.99 and p .007

Table 3: Cytotrophoblastic proliferation

Cytotrophoblastic proliferation	Control		IUGR	
	No. of cases	Percentage	No. of cases	Percentage
<10	108	54%	74	37%
10-20	66	33%	83	41.5%
>20	26	13%	43	21.5%
Total	200	100%	200	100%

Chi square value 12.5 and p .002

Table 4: Number of areas of fibrinoid necrosis in villi

Number of area fibrinoid necrosis in villi	Control		IUGR	
	No. of cases	Percentage	No. of cases	Percentage
<5	171	85.5%	143	71.5%
>5	29	14.5%	57	28.5%
Total	200	100%	200	100%

Chi square value 11.6 and p .001

Table 5: Number of Areas of hylinized villi

Number of Area hylinized villi	Control		IUGR	
	No. of cases	Percentage	No. of cases	Percentage
<5	132	66%	99	49.5%
>5	68	34%	101	50.5%
Total	200	100%	200	100%

Discussion

Placenta being a fetal organ has a similar anxiety, to which the baby is uncovered. Subsequently any infection cycle influencing the mother and hatchling additionally enormously affects placenta. Ordinarily the placental morphology differs extensively during its short life span. [5]

Modifications in placenta as a feature of "Maturing" marvel are most likely a piece of development measure and go connected at the hip with proceeded with development of placenta. Placenta develops till 37th week and therefore youthful villi are seen even till term. Thus in the examination on placenta Fox (1975) has focused on the significance of investigating the placental pathology quantitatively and has expressed that the significance of the injuries could be acknowledged just when surveyed in connection of fetal development and development.

The histology of placenta of moms of IUGR patients likewise shows huge expansion in syncytial tie development, cytotrophoblastic cell multiplication, stromal fibrosis, calcification and hyalinisation of villi in contrast with the control group. [6] Exceptionally huge expansion in the rate of dead tissue, intervillous fibrin testimony, stromal fibrosis and syncytial hitching were found in IUGR placentas contrasted with full term typical placentas on tiny examination. [7,8]

They additionally found as non-huge expansion in cytotrophoblastic cell multiplication. Syncytial hitches development, cytotrophoblastic cell multiplication, stromal fibrosis, calcification and hyalinisation of villi is up managed by intra placental hypoxia and down controlled by expanding intra placental oxygen levels. [9] This likewise demonstrates a degenerative cycle as reaction to nearby hypoxia. This is viewed as a versatile marvel to decrease the dispersion distance from the intevillous space to the fetal vessels within the sight of diminished oxygen pressure. [10,11]

Conclusion

Histopathological findings in low power field like syncytial knot formation, cytotrophoblastic cellular proliferation, calcification and hyalinisation of villi are also found as normal aging changes in placenta but it occur early and more frequently per low power field in IUGR group. To conclude, these morphological & histological findings of placenta are the etiological basis for Intra uterine growth restriction.

References

1. Pardi G, Marconi AM, Cetin I. Placental-fetal interrelationship in IUGR fetuses: a review. Placenta. 2002; 23:136-41.
2. Mardi K, Sharma J. Histopathological evaluation of placentas in IUGR pregnancies. Indian J Pathol Microbiol. 2003; 46(4):551-4.
3. Curtin WM, Krauss S, Metlay LA, Katzman PJ. Pathologic examination of the placenta and observed practice. Obstet Gynecol. 2007J; 109(1):35-41.
4. Tantbirojn P, Saleemuddin A, Sirois K, Crum CP, Boyd TK, Tworoger S, et al. Gross abnormalities of the umbilical cord: related placental histology and clinical significance. Placenta. 2009; 30:1083-8.
5. Disalvo DN. Sonographic evaluation of the placental cord insertion site. AJR Am J Roentgenol. 1998; 170(5):1292–8.
6. Rani N. Structural changes in placentas of preeclamptic patients. J Anatomical Soc India. 2009; 59(1):48–139.
7. Hemlata M, Kumar MP, Jankai M, Dudala SR. Histopathological evaluation of placentas in IUGR pregnancies. Indian J Pathol Microbiol. 2014; 1(4):566–9.
8. Kotgirwar S, Ambiye M, Athavale S, Gupta V, Trivedi S. Study of Gross and Histological features of placenta in intrauterine growth retardation. J Anat Soc. 2011; 60(1):37–40.
9. Biswas S, Ghosh SK. Gross morphological changes of placentas associated with intrauterine growth restriction of fetuses: a case control study. Early Human Develop. 2008; 84(6): 357–62.
10. Dhabhai P, Gupta G. Placental weight and surface area in iugr cases. Innov J Med Health Sci. 2014; 4(6):198–200.
11. Meèëjus G. Influence of placental size and gross abnormalities on intrauterine growth retardation in high-risk pregnancies. Acta Med Litu. 2005; 12(2):14–9.