

Original Research Article

A Study of Correlation of Changes in Placental Anatomy in Pregnancy Associated with IUGR (Intra Uterine Growth Restriction)

Dr. Prof Sumitra Yadav¹, Dr. Shweta Pradhan¹, Dr. N. Srivastav²¹Dept of Obstetrics & Gynecology, MGMMC & MY Hospital, Indore²Dept of Anatomy, MGMMC & MY Hospital, Indore

*Corresponding author

Dr Shweta Pradhan

Email: shwetapradhan22@gmail.com

Abstract: Placenta is a vital organ for maintaining pregnancy and promoting normal growth by transfer of essential nutrients between fetus and mother. Any morphological alteration of the placenta affects the growth of fetus leading to intrauterine growth restriction (IUGR). The purpose of this study was to find out morphological and histopathological changes of placenta and to evaluate correlation of IUGR and placental histology. This study included 400 pregnant patients whose fetuses had intrauterine growth restriction and 200 patients with normal foetal growth. Gross and histopathological features of placentas of both groups were studied, analysed by student's t test and compared with chi square test. p values of < 0.05 were considered significant. Gross features, weight, thickness and calcification in study group show significant increase in value (p < 0.05) compared to control group and histopathology of placenta in study group also shows significant increase in syncytial knots formation, cytotrophoblastic proliferation, stromal fibrosis and calcification as compared to control group. To conclude, whatever may be the cause of IUGR, these morphological and histopathological changes lead to decrease blood to the placenta and this restriction of blood flow ultimately causes IUGR in the foetus.

Keywords: Placenta, I.U.G.R., Syncytial Knot, Cytotrophoblastic Proliferation.

INTRODUCTION

IUGR is observed in about 23.8% of the new born and approximately 30 million babies suffer from IUGR every year. WHO report 2010 found the prevalence of low birth weight baby in India to be 26%. The proportion of IUGR was found to be 54.2% in India (Deonis M .Blossener). IUGR is associated with smaller than normal placenta and there is increased perinatal mortality and morbidity. It's the placenta not the fetus that is initially affected. Despite being the most readily available surgical sample placenta happens to be the least well studied one, and neither its anatomy nor pathology are well understood on the account of its rapid growth short life span and quick senescence. Present study has been undertaken to compare the morphology and histology of normal placentae with the IUGR placentae and correlate this findings with the birth weights of newborns.

AIMS and OBJECTIVES

This study is done to evaluate anatomical and histopathological changes in placenta of IUGR patients and further to evaluate correlation of IUGR with placental anatomy and histology.

MATERIALS AND METHODS

The study was conducted in the department of obstetrics and gynecology from May 2012 to Sept 2016 on 400 patients.

Selection criteria

Inclusion criteria

- Patients with baby weight <10 th percentile, IUGR due to all causes.
- Patient who gave informed consent.

Exclusion criteria

- Preterm patients

- Damaged placenta
- Lack of consent.

Study design

All the patients in the study are hospitalized. Placenta and cord with membranes were collected immediately after delivery and were preserved in 10% formalin. Fetal weight and outcome were recorded immediately after birth. Placenta was examined for its color, weight, diameter, thickness, and number of cotyledons infarcted, and calcified area. The cord was

examined for position of insertion and abnormality. Histological processing was done under fixation, dehydration, clearing, and paraffin embedding block preparation, cutting and staining. Stains used were haematoxylin, periodic acid Schiff and masson's trichrome. Special attention was paid to the cells, stroma, and blood vessels in the chorionic plates, basal plate and terminal villi.

OBSERVATIONS AND RESULTS

Table-1: Distribution of Age

AGE (years)	Control		IUGR	
	No.	%	No.	%
<20	11	5.5	13	6.5
20-30	184	92	179	89.5
>30	5	2.5	8	4
TOTAL	200	100	200	100

Table2: Mode of delivery

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

Weight of placenta

Weight	Control		IUGR	
	No.	%	No.	%
250-300	0	0	10	5
301-350	0	0	60	30
351-400	0	0	105	52.5
401-450	20	10	30	15
451-500	60	30	5	2.5
501-550	110	55	0	0
551-600	10	5	0	0
Total	200	100	200	100

Volume of placenta

Vol(ml)	Control	IUGR
Mean volume	495.12	330.45

Diameter of Placenta

Diameter(cm)	Control	IUGR
Mean diameter	25.8	20.61

(p<0.05)

Thickness of placenta

Thickness (cm)	Control	IUGR
Mean Thickness	5.06	4.01

Weight of the Placenta: Birth Weight of Baby (Mean Placental co-efficient)

Weight ratio	Control	IUGR
Mean	0.1921	0.1921

(p<0.05)

No. of Cotyledons

Cotyledons	Control		IUGR	
	No.	%	No.	%
<15	18	9	21	10.5
15-18	162	81	179	89.5
>18	20	10	7	3.5
Total	200	100	200	100

(Chi Square value 2.257 and p value 0.3235)

Insertion of cord

Insertion of cords	Control		IUGR	
	No.	%	No.	%
Concentric	154	77	122	61
Eccentric	36	18	67	33.5
Velamentous	6	3	5	2.5
Marginal	4	2	6	3
Total	200	100	200	100

Infarcted Placenta

Infarcted Placenta	Control		IUGR	
	No.	%	No.	%
Present	52	26	74	37
Absent	148	74	126	63
Total	200	100	200	100

Placental maturity

Placental maturity	Control		IUGR	
	No.	%	No.	%
0	0	0	0	0
I	7	3.5	1	0.5
II	127	63.5	98	49
III	66	33	101	51
Total	200	100	200	100

No of Calcified Areas

Calcific areas	Control		IUGR	
	No.	%	No.	%
<5	146	73	39	19.5
5-25	46	23	114	57
>25	8	4	47	23.5
Total	200	100	200	100

Number of syncytial Knot Formation

Syncytial Knot Formation	Control		IUGR	
	No.	%	No.	%
<10	42	21	31	15.5
10-25	152	76	158	79
>25	01	0.5	11	5.5
Total	200	100	200	100

Cytotrophoblastic proliferation

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

No of Areas of fibrinoid necrosis in villi

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

No of stromal Calcification

Cytotrophoblastic proliferation	Control		IUGR	
	No.	%	No.	%
<5	176	88	48	24
>5	24	12	152	76
Total	200	100	200	100

Chi square value 166 and p .0001

Number Of areas of Stromal Fibrosis

Number Of stromal fibrosis areas /LPF	Control		IUGR	
	No.	%	No.	%
<5	196	98	43	21.5
>5	4	2	157	78.5
Total	200	100	200	100

Chi square value 73.3 & p 0.0001

No of areas of stromal Hyalinization

No of stromal hyalinization areas	Control		IUGR	
	No.	%	No.	%
<5	128	64	96	48
>5	72	36	104	52
Total	200	100	200	100

The gross anatomic features of placenta eg. Calcified areas and marginal insertion of the umbilical cord in the study group show significant increase in value when compared to control group.

The histology of placenta of mothers of IUGR patients also show significant increase in syncytial knot formation, cytotrophoblastic cellular proliferation, stromal fibrosis, calcification and hyalinization of villi and stroma in comparison to the control group.

The study confirms the risk of caesarean delivery is increased in IUGR group. The study also reveals that the ratio of weight of the placenta to new born is increased IUGR and placental maturity (by ultrasonography) is more in IUGR.

DISCUSSION

There is great deal of debate going on whether placental inefficiency is a primary cause, contributory factor or a result of extraneous factors (infection, genetic, nutritional, etc.) in cases of IUGR [1,2]. Placental insufficiency, in some form or fashion, is associated with the majority of cases of IUGR. The placental dimensions found in the present study were significantly reduced as compared to the control group. Similar findings have been reported by Malik *et al.* [3]. Other workers have not considered these criteria in their studies. Fetal and placental weights were significantly reduced and were similar to findings reported in previous studies [3, 2, 4]. Fibrinoid necrosis is well recognized as one of the hallmarks of immune attack on trophoblastic cell. The fibrinoid material in the affected villi contains a considerable quantity of immunoglobulins. Fibrinoid necrosis was observed in 28% cases in our study as compared to 32%-38% cases as reported by the other workers [3,4]. This difference may be attributed to selection of IUGR cases. In the present study we considered all causes of IUGR while others included only idiopathic IUGR.

CONCLUSION

Normally the placental morphology varies considerably during its short life span. Alteration in placenta as a part of ageing phenomenon are probably a part of maturation process and go hand in hand with continued growth of placenta. The placenta of IUGR ages earlier than control group. Ratio of weight of placenta to new born is increased in IUGR group. Morphological findings like lowweight placenta,

marginal insertion of cord, etc. are also found in normal placenta but more frequent in IUGR group.

To conclude, these morphological and histological findings of placenta are the etiological basis for intra uterine growth restriction.

REFERENCES

1. Cetin I, Alvino G. Intrauterine Growth Restriction: Implications for placental metabolism and transport-A review Placenta.2009;(30):77-82
2. Althshuler G, Russell P, Ermochilla R. The placental pathology of small for gestational age infants. Am J obstet. Gynecol.1975; 121: 351-59
3. Malik SR, Gupta AK, Choudhry S. Ocular cysticercosis. American journal of ophthalmology. 1968 Dec 1;66(6):1168-71.
4. Bhatia AB. Ultrasonic absorption. Clarendon Press; 1967.