



International Journal of Medical Research & Health Sciences

www.ijmrhs.com

Volume 4 Issue 2

Codon: IJMRHS

Copyright @2014

ISSN: 2319-5886

Received: 2nd Dec 2014Revised: 6th Jan 2015Accepted: 16th Jan 2015

Research article

INFARCTION IN NORMAL AND INTRAUTERINE GROWTH RETARDATION [IUGR] PLACENTA

*Pooja Dhabhai¹, Ghanshyam Gupta²^{1,2}Department of Anatomy, R.N.T. Medical College, Udaipur, Rajasthan, India

*Corresponding author email: poojadhabhai14@gmail.com

ABSTRACT

Background and Purpose: The purpose of the study is to compare the presence of Infarction in normal placentas and IUGR placentas. **Study design and setting:** Research study, Department of Anatomy, R.N.T. Medical College, Udaipur. **Study Sample:** 100 control and 100 IUGR Placentas **Inclusion criteria:** 100 Placentas from normal control Pregnancies and 100 Placentas from mother who Delivered Intra Uterine Growth Retarded (IUGR) babies **Exclusion Criteria:** we refer only uncomplicated Pregnancies without any previous diseases **Results:** Chi Square test was used for statistical analysis. **Conclusion:** Increased incidence of extensive infarction associated with low fetal weight

Keywords: Infarction, Placenta, Intrauterine Growth Retardation

INTRODUCTION

Fetal growth depends on the proper development and function of the placenta, which serves to maintain mater no fetal interference for the exchange of blood gases, nutrients, and waste^[1]. The architecture of the placenta is altered in many maternal diseases such as diabetes mellitus^[2], hypertension^[3], preeclampsia [PE]^[4], and eclampsia^[5]. Although the placenta is a vital organ, its systemic study has been neglected; however, in recent times, it has evoked great interest, and much work is being conducted to understand the unique biological status of this complex organ^[6]. Placental examination has clinical value in cases of PE and intrauterine growth retardation (IUGR), both of which are associated with high perinatal morbidity and mortality accompanied with gross pathological changes in the placenta.

Placental infarcts are usually wedge shaped and always have a point of contact with the basal plate, when fresh they are well demarcated, dark red and moderately firm^[7]. Placental infarctions are zone of

ischaemic necrosis of group villi due to complete interference with their blood supply in the deciduas or in the local state by thrombosis of a spiral arteriole or a retroplacental haemorrhage^[8]. Small areas of infarction, involving less than 5% of the parenchyma, were found in almost a quarter of placentas from normal pregnancies and are of no clinical significance. Extensive infarction, that is involving more than 10% of villous substance is associated with a high incidence of fetal hypoxia, low birth weight and fetal death and is virtually confined to placentas from patients suffering from the hypertensive complications of pregnancy. Extensive infarction is due to occlusion of multiple maternal arterioles^[7]. It was found that extensive infarction in cases of toxemia were associated with low birth weight, placental weight and increased foetal death^[7]

MATERIAL AND METHODS

The study of placenta in normal and IUGR cases was carried out at R.N.T. Medical College & Hospital, Udaipur. The cases were studied from 1-7-13 to 1-5-14. The study plan was approved by institution Ethical Board and consent form was filled by patients.

The placenta were collected from 200 women admitted to the labour Rooms of the hospital (either directly or through the antenatal wards). All the cases were within the age group of 18-40 years, of average height and weight. Group 1-normal pregnancy 100 patients included in this group, normal Hb and urine analysis, not associated with any disease.

Group 2-IUGR cases 100 cases of IUGR were included. After the delivery placenta were collected for gross studies, washed and surface dried between blotting papers. Presence of Infarction noted as Mild (less than 5% of total placental area) Moderate (more than 5% less than 10% of total placental area) Severe (more than 10% of total placental area) [7]

Area of infarction on the maternal surface varied from no Infarcted area to 5-10 % of the total surface (as calculated from combined area of the infarcts as seen on the maternal surface.) [7]

RESULTS

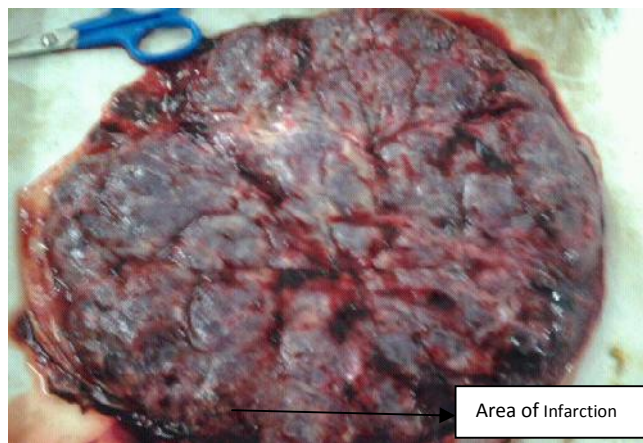


Fig.1 Photograph of maternal surface of placenta showing area of Infarction

Table 1 Analysis for Infarction

Infarction type	Normal pregnancies group (n = 100)	IUGR pregnancies group (n = 100)	p value
Nil	87	4	<0.0001 *

Mild (less than 5% of total placental area)	9	51	
Mod (more than 5% less than 10% of total placental area)	4	20	
Severe (more than 10% of total placental area)	0	24	

*Highly significant $p < 0.0001$

Table 2. Statistical comparison of Infarction present in control and research group

Author	Place	No. of cases	Infarction present in % of cases		Result
			Control	Research	
Ganga R Singal (2013) ⁹	Bhavnagar	100	5	10	<0.01
Kotgirwar (2011) ¹⁰	Bhopal	55	nil	1.8	<0.01
Pradeep S Londhe ¹¹	Andhra Pradesh	374	5.4	10.6	<0.01
Figen Barut ¹²	Turkey	110	nil	92.7	<0.01
Gediminas Meëjus ¹³	Lithuania	120	4.2	49.2	<0.01
Nayereh Ghomian ¹⁴	Iran	46	8.7	39.1	<0.0001
Günyeli ¹⁵	Turkey	52	4	58	<0.05
Present Study	India-Udaipur	200	13	96	<0.0001

*Highly significant $p < 0.0001$, *Significant $p < 0.01$, $p < 0.05$

DISCUSSION

Present study shows that infarction is present in higher % of cases in IUGR group and the difference is highly significant in our study. The p value (< 0.0001) is highly significant. The present study is consistent with Nayereh Ghomian et al¹⁴ also shows Highly significant values of infarction in research group.

Among Indian studies the present study is consistent with study of Ganga R Singal⁹, Kotgirwar¹⁰. Pradeep S Londhe¹¹ also studied higher percentage of infarction in research group. The p value (< 0.01) is significant and thus favours the present study. Among western studies the present study is consistent with Figen Barut¹², Gediminas Meëju¹³, Günyeli et al¹⁵ as they also showed higher occurrence of infarction in IUGR group. In present study infarction was seen in 13 cases of normal terms pregnancy but

extent of infarction was less than 10% of placental tissue. It was seen in 96% cases of IUGR, in 24% of these extent of infarction was more than 10% of placental tissue on gross examination.

CONCLUSION

Increased incidence of extensive infarction was seen in cases of IUGR. These cases were associated with low foetal weight. Every placenta shows many degenerative features. Presumably these are to an extent, physiologic sequence of evolution. However, when they occur in excess, they must be considered as pathological, particularly when they affect foetal growth deleteriously.

ACKNOWLEDGEMENT

Conflict of Interest-NIL

REFERENCES

1. Vogel P. The current molecular phylogeny of Eutherian mammals challenges previous interpretations of placental evolution. *Placenta*. 2005; 26:591–96.
2. Pardo F, Arroyo P, Salomón C, Westermeier F, Guzmán-Gutiérrez E, Leiva A, Sobrevia L. Gestational diabetes mellitus and the role of adenosine in the human placental endothelium and central nervous system. *J Diabetes Metab*. 2012; 2:10-11.
3. Barker DJ, Bagby SP, Hanson MA. Mechanisms of disease: in utero programming in the pathogenesis of hypertension. *Nat Clin Pract Nephrol*. 2006;2:700–07.
4. Sankar KD, Bhanu PS, Kiran S, Ramakrishna BA, Shanthi V. Vasculosyncytial membrane in relation to syncytial knots complicates the placenta in preeclampsia: a histomorphometrical study. *Anat Cell Biol*. 2012; 45:86–91.
5. Akhlaq M, Nagi AH, Yousaf AW. Placental morphology in pre-eclampsia and eclampsia and the likely role of NK cells. *Indian J Pathol Microbiol*. 2012; 55: 17–21.
6. Murphy VE, Smith R, Giles WB, Clifton VL. Endocrine regulation of human fetal growth: the role of the mother, placenta, and fetus. *Endocr Rev*. 2006; 27: 141–69.
7. Fox, H. in *Post graduate obstetrical and Gynecological Pathology* by Fox, H and Langley, F. a. 1st Ed. 1973; 409-37,
8. Zeek PM, Assali NS. Vascular changes in the deciduas associated with eclamptogenic toxemia of pregnancy. *American Journal of clinical Pathology*, 1950; 20: 1099-09.
9. Dr. Ganaga R, Singal et al, Placental Morphometry in Relation to Birth Weight of Full Term Newborn; *SEAJCRR* 2013; 2(5) 334-42
10. S kotgirwar, M ambiye, S athavale, V gupta, S trivedi, Study of Gross and Histological features of placenta in intrauterine growth retardation; *J. Anat. Soc. India* 2011 60(1) 37-40
11. Londhe, Pradeep S. et al Placental morphometry in relation to birth weight of full term newborn babies. *National journal of integrated research in medicine*. 2012; 3(1): 67-72.
12. Figen Barut et al; Intrauterine growth restriction and placental angiogenesis; *Diagnostic Pathology* 2010; 5: 24
13. Gediminas meëėjus, Influence of placental size and gross abnormalities on intrauterine growth retardation in high-risk pregnancies, *Acta medica lituanica*. 2005; 12 (2) 14–19
14. Ghomian, Nayereh et al 2014 *Iranian Journal of Pathology*; Winter 2014; 9 (1): 9
15. Günyeli et al. Placental examination in IUGR and Stillbirth; *J Turkish-German Gynecol Assoc* 2011; 12: 75-9