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Study of Placental Insufficiencies in IUGR Babies in Term Pregnancies

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Abstract

Introduction: In intrauterine growth restricted babies, at term, the placenta might hold the key to the etiology. This present study is aimed at comparison of data of gross and pathological study of placentae from normal weight (control) and IUGR groups and attempted to establish a relationship between placenta pathology and intrauterine growth retardation in term pregnancy. **Aims and Objectives:** 1. To study placental factors associated with IUGR. 2. To study prevalence of placental factors in details. **Material and Methods:** A study of 100 patients, with more 37 and less 42 weeks of gestation wasconducted for 2yrs in a tertiary care hospital. A thorough general examination & Systemic examination which included per abdominal examination including uterine height, abdominal girth, symphysio fundal height, estimated fetal weight, abdominal grips, fetal heart auscultation, and per vaginal examination. Routine and specific investigations will be sent for clinical correlation. Placenta obtained after delivery was send for macroscopic and microscopic examination to pathologist. The reports obtained will be used to study placental causes in IUGR and placenta in detail. **Result:** Average placental weight of term pregnancy is 400 gms and placental coefficient is 0.18. decidual surface area is 254 cm^2 and its thickness is 2.72cm. Retroplacental haemorrhage is closely associated with PIH. Microscopic infarction is found with high frequency ($p<0.005$). Placenta of IUGR fetus, gross pathological changes like severe infarction with or without retroplacental haematoma can obviously be noted but they are not found to be statistically significant. Placental coefficient is increased in cases of anemia. Syncytial knot formation in excess and thickening of basement membrane is well correlated.

Keywords: Placental Insufficiency, IUGR

1. Introduction

Placenta is one of the most challenging organs; its function often holds the key to fetal development. It is an instrument of transfer of essential elements i.e nutrients and oxygen from mother to embryo and the waste products of metabolism from embryo to mother.

Low Birth Weight (LBW) is one of the most important causes of neonatal loss. Low birth weight is defined as one whose birth weight is less than 2500gm, irrespective of gestational age (international classification of diseases of world health organization, 1977). Intrauterine Growth Restriction (IUGR) or Small For Date (SFD) babies are those whose birth weights are disproportionately low for gestational age (<10^{th} percentile) or 2 SD.

In intrauterine growth restricted babies, at term, the placenta might hold the key to the etiology. Though the contribution of placental changes remained controversial, it was accepted that IUGR was associated with fetal hypoxia resulting partially from alteration in growth and development of placental villi and their vasculature.

This present study is aimed at comparison of data of gross and pathological study of placentae from normal weight (control) and IUGR groups and attempted to establish a relationship between placenta pathology and intrauterine growth retardation interm pregnancy.

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2. Aims and Objectives

1. To study placental factors associated with IUGR.
2. To study prevalence of placental factors in details

3. Material and Methods

3.1 Study Design

- Type of study: observational
  -Study setting: Tertiary health centre, Nashik
  -Study duration: 2 years (Aug 2011- Aug 2013)
  -Study participants:
- Sample size: 100 cases
- Inclusion criteria:
  -All term deliveries >37 weeks and <42 weeks included in the study irrespective of age, parity and mode of delivery from 2011 to 2013.
  -Irrespective of obstetric complications like PIH, anemia, cardiac disease.
- Exclusion criteria:
  -Multiple gestations
  -Any previous history of congenital anomaly.

4. Methodology

The present study was conducted in Tertiary health care centre Nashik. A total of 100 participants after satisfying inclusion and exclusion criteria were included.Written informed consent were obtained from the patients. A proforma was used to collect medical history and clinical findings of patient.

A study of 100 patients, with more than 37 weeks and less than 42 weeks of gestation, who present to the tertiary health centre in the department of obstetrics and gynecology was conducted for 2 years.

Cases were consisting of patient referred to us from our hospital and also patient referred from outside to our hospital for delivery.

History

A detailed history of the patient was recorded, that included her history and period of gestation calculated from last menstrual period and confirmed by ultra sonography. Information regarding systemic illness was obtained and past obstetrics and medical records were analyzed. Past history of hospitalization or any major illness is also included.

A thorough general examination of the patient was carried out wherein the height, weight blood pressure, pulse rate and respiratory rate, pallor, oedema, lymphadenopathy, cyanosis, icterus was recorded. A systemic check up including the cardiovascular, respiratory, and gastrointestinal and central nervous system was carried out.

Systemic examination included thorough per abdominal examination including uterine height, abdominal girth, symphysiofundal height, estimated fetal weight, abdominal grips, fetal heart auscultation, and per vaginal examination. Routine and specific investigations were sent for clinical correlation. Placenta obtained after delivery was send for macroscopic and microscopic examination to pathologist. The reports obtained were used to study placental causes in IUGR.

5. Results

PIH

Present study included 23 cases of PIH. They are classified as mild and severe. Cases of eclampsia were included in severe PIH (9 severe and 14 mild). Gross features of placenta were compared to other studies (Table 1).

a. Extent of Infarction

In mild PIH cases placental infarction was not extensive and not involved more than 5% of placental tissue. In placenta from severe PIH cases incidence as well as extent was more. Findings were correlating with above studies (Table 2).

Table 1. Microscopic placental lesions in PIH

Description	No of cases n=23	Microscopic placental lesions in PIH (%)
Infarction	12	52%
Calcification	16	70%
Perivillous fibrin deposit	0	0%
Hyaline changes	0	0%
Villous change	0	0%
Increased syncytial knot	6	26%
Cytotrophoblastic proliferation	4	17%
Basement membrane thick	5	22%
Villous fibrosis	0	0%
Fibrinoid necrosis of villi	2	9%
Lymphocytic infiltration	2	9%
Membrane changes	0	0%

Small haematomas in a complicated and a large haematoma in uncomplicated pregnancy will cause fetal distress (Table 3).

Currently attention is given to villous morphological changes in form of increased syncytial knot, cytotrophoblast proliferation, basement membrane thickening, and stromal fibrosis (Table 4).

Observation in this study were comparable to Sodhi et al.[6] cytotrophoblastic proliferation may be a response to chronic placental ischemia and these cells perhaps secrete basement membrane substance to cause high basement thickening. Stromal fibrosis and increased syncytial knot have been attributed to decreased fetal perfusion.

Mean birth weight in PIH was found to be 2132.6 gms (+-528.9) in term pregnancies as compared to normal pregnancies 2774.2 gms (+- 338). Mean placental coefficient was 0.182.

IUGR

IUGR was studied in 100 cases which were categorized with associated maternal disorders (Table 5)

a. Associated with PIH
b. Associated with anemia
c. Only IUGR

In the first group there were 23 cases (23%). Association of IUGR with PIH is well known and most common cause.

Table 2. Extent of infarction

Extent	Wentworth[1]	Fox[2]	present
mild	11.915	34%	23%
severe	67%	60%	50%
normal	20%	25%	21.9%

Table 3. Extent of Basal decidual haematoma

Extent	Wentworth[1]	Fox[2]	present
Mild	78%	12-15%	21.75%
severe	25%		

8 cases were associated with anemia. The placental coefficient was found to be 0.27. The only consistent finding was increased syncytial knot with few gross anomalies.

38% of placenta was not associated with any abnormality except IUGR were found to have grossly infracted lesions. Three placentae showed 2/3 of mass i.e old infarction. Histologically they showed syncytial knot formation and stromal fibrosis of villi.

Anemia

The relationship between maternal haemoglobin level was measured by Sahli's method. Macroscopic and histological features of placenta were studied in 8 cases (Table 6). The presence of anemia was associated with higher placental weight, lower fetal weight, better placental correlation, increased villous fibrosis, perivillous fibrin deposition, increased syncytial knot and basement

Table 5. Microscopic changes in placenta of IUGR

Description	No of cases n=38	Microscopic changes in placenta of IUGR (%)
Infarction	26	68%
Calcification	20	53%
Perivillous fibrin deposition	5	13%
Hyaline changes	0	0%
Villous morphology		0%
Increased syncytial knot	9	24%
Cytotrophoblastic proliferation	7	18%
Basement membrane thick	13	34%
Villous fibrosis	0	0%
Villous oedema	0	0%
Fibrinoid necrosis of villi	3	8%
Vascular changes	14	37%
Lymphocytic infiltration	0	0%

Table 4. Villous morphological changes in PIH cases

Villous lesion	Sodhi[6]		Present	
	mild	severe	mild	severe
Increased syncytial knot	17-48	19-49	43	60
Cytotrophoblastic proliferation	10-40	20-35	6-25	30
Basement thickening	1-6	1-14	6-25	50
Stromal fibrosis	3-10	6-15	0	10

Table 6. Microscopic changes in anemia

Description	No of cases n=8	Microscopic changes in anemia (%)
Infarction	5	63%
Calcification	6	75%
Perivillous fibrin deposition	2	25%
Hyaline change	0	0%
Villous change		0%
Increased syncytial knot	1	13%
Cytotrophoblastic proliferation	1	13%
Basement membrane thick	3	38%
Villous fibrosis	0	0%
Villous oedema	0	0%
Fibrinoid necrosis of villi	1	13%
Vascular changes	3	38%
lymphocytic infilteration	3	38%

membrane thickening. Calcification was also more prominent in gross as well as microscopic examination. These changes may be the result of maternal undernutrition[3] or it may be due to hyopoxia caused by anemia, when severe.

These finding were consistent with those of other workers (Rusia, mukherjee and Mitra)[4,5] placental coefficient in this cases was 0.27. it was similar to that from Beischer[8]. Perinatal outcome in this study was increased in IUGR(30%).

6. Summary

One hundred women with normal and abnormal pregnancy were studied. The gross and microscopic pathological features of placenta and fetal weight were correlated. Women had mean parameters like age 23.28(+-319) years, gravidity 2.05 (+-1.13), gestational age 37.25 (+-3.79) and haemoglobin 9.5gm% (1.27).

1. Average placental weight of term pregnancy was 400 gms and placental coefficient is 0.18. decidual surface area was 254 cm^2 and its thickness was 2.72cm
2. Frequent but insignificant correlation of gross infarction is found in placenta from PIH. Retroplacental haemorrhage was closely associated with PIH. Microscopic infarction was found with high frequency ($p<0.005$). Increased syncytial knot, fibrinoid necrosis was of villi, and cytotrophoblastic proliferations were strongly associated with PIH. Mean birth weight from these patients was less, compared to normal pregnancy but placental coefficient was not much affected.
3. From the study of placenta of IUGR fetus, gross pathological changes like severe infarction with or without retroplacental haematoma can obviously be noted but they were not found to be statistically significant. Microscopic changes i.e basement membrane thickening and cytotrophoblastic proliferation were noted in excess and suggestive of decreased fetoplacental perfusion due to vascular lesion. Average placental weight, thickness and decidual placental area were significantly less in IUGR. They were strongly correlated to IUGR.
4. Placental coefficient was increased in cases of anemia. Syncytial knot formation was in excess and thickening of basement membrane was well correlated.

7. References

1. Wentworth P. Placental infarction and toxemia of pregnancy : Am J Obstet Gynecol. 1967;99:318.
2. Fox H. Haines and Taylor Obstetrical and Gynecological Pathology. 3rd edn. Churchill Livingstone, Edinburgh, London, Melbourne and New York; 1987.
3. Khanna S, Agarwal KN, Murthy LS. Placental histological changes in maternal undernutrition. Ind J Med Res. 1977; 66:429.
4. Russell P. Inflammatory lesions of the human placenta III – The histopathology of villitis of unknown etiology. Ostet Gynecol Survey. 1981; 36:362.
5. Mukherjee NK, Mitra NK. The effect of malnutrition on placenta. Ind J Pathol Microbiol. 33(4):1990; 314–322.
6. Sodhi S. Placental pathology in pre-ecclampsia ecclampsia syndrome. Ind J Pathol Microbiol. 1990 Jan; 33(1):11–6.