ABSTRACT

INTRODUCTION – IUGR is the leading cause of fetal mortality and morbidity. Although the etiology of IUGR remains unknown, placental and umbilical cord diseases are clinically the most relevant. Thus, placental and umbilical cord examination in IUGR cases provide a specific pathophysiological explanation that may recur in subsequent pregnancies and lead to change in follow up and management.

AIM - To study the placent al and umbilical cord pathology in case of intrauterine growth retardation.

MATERIAL AND METHODS – During the study period July 2012 to June 2014, after delivery of the intrauterine growth retardation cases admitted in Obstetrics ward of Govt Medical College and ass. Dr Susheela Tiwari Govt hospital, Haldwani, Nainital,Uttarakhand, placenta and umbilical was collected and its clinical and pathological examination was done.

RESULT – In this Prospective clinico pathological study, findings in 78 cases with IUGR were reviewed and compared with 84 normal control cases. The incidence of IUGR is significantly high in unbooked antenatal cases. Grossly the average placental weight ,size and placental ratio is significantly low in IUGR group. Microscopically increase ST knots(65.38%), Decidual vasculopathy(33.33%), villous infarction (38.6%), villous fibrosis (38.46%), intravillous thrombus (25.64%), syncitial knots(65.38%) were significantly high in IUGR group. Mean cord length is low in IUGR group. Grossly true knot and cord around neck and microscopically endovasculitis(20%), thrombosis(33.33%) were significantly associated with IUGR group.

CONCLUSION - Placental and umbilical cord abnormalities are significantly associated with IUGR and may be a major factor in the pathogenesis of abnormal fetal growth.

KEYWORDS – IUGR, Placental and umbilical cord abnormalities, placental infarction

1. INTRODUCTION

Fetal growth in utero is dependent on genetic, placental and maternal factors. The fetus is thought to have an inherent growth potential that, under normal circumstances, yields a healthy newborn of appropriate size. The maternal-placental-fetal units act in harmony to provide the needs of the fetus while supporting the physiologic changes of the mother. Limitation of growth potential in the fetus is analogous to failure to thrive in the infant. The causes can be intrinsic or environmental. Fetal growth restriction is the second leading cause of perinatal morbidity and mortality, followed only by prematurity. The incidence of intrauterine growth restriction (IUGR) is estimated to be approximately 5 percent in the general obstetric population. The present study aims at evaluating the placental and umbilical cord pathological changes associated with intrauterine growth restriction. Placental and umbilical cord examination in intrauterine growth retardation cases provide a specific pathophysiological explanation that may recur in subsequent pregnancies and lead to change in follow up and management.

AIM AND OBJECTIVES

- To study the various gross and microscopic findings in placenta and umbilical cord of intrauterine growth restriction.
- To correlates placental and umbilical cord finding with fetal and neonatal outcome.

DEFINITION, TYPES AND CAUSES OF IUGR

The classic definition of intrauterine growth retardation refers to “an infant with a birthweight equal to or less than the 10th percentile for gestational age” 10 or to an infant weighing less than 90% of the population for gestational age. IUGR is usually classified as symmetric and asymmetric.
1) **Reduced growth potential** (formerly symmetrical intrauterine growth retardation) implies a fetus whose entire body is proportionally small.

2) **Nutritional** intrauterine growth retardation (formerly asymmetrical intrauterine growth retardation) implies a fetus who is undernourished and is directing most of its energy to maintaining growth of vital organs, such as the brain and heart, at the expense of the liver, muscle and fat. This type of growth restriction is usually the result of placental insufficiency.¹

IUGR is a complicated placental vascular disease resulting in low birth weight, preterm delivery, and increased perinatal morbidity and mortality.¹²³ Four to six percent of all births in the United States are IUGR.³⁴ IUGR may be caused by various fetal, maternal, and placental factors.

**Causes of Intrauterine Growth Restriction**⁷

1). Placental insufficiency
- Unexplained elevated maternal alpha-fetoprotein level
- Idiopathic
- Preeclampsia

2). Chronic maternal disease
- Cardiovascular disease
- Diabetes
- Hypertension

3). Abnormal placentation
- Abruptio placentae
- Placenta previa
- Infarction
- Circumvallate placenta
- Placenta accreta
- Hemangioma

4). Genetic disorders
- Family history
- Trisomy 13, 18 and 21
- Triploidy
- Turner's syndrome (some cases)

5). Malformations

6). Immunologic
- Antiphospholipid syndrome

7). Infections
- Cytomegalovirus
- Rubella
- Herpes
- Toxoplasmosis

8). Metabolic
- Phenylketonuria
- Poor maternal nutrition

9). Substance abuse (smoking, alcohol, drugs)

10). Multiple gestation

11). Low socioeconomic status

**II. MATERIAL AND METHODS**

The study was conducted in the department of Obstetrics and Gynaecology, Government Medical College and associated Dr. Sushila Tiwari Government Hospital, Haldwani between July 2012 to June 2014. The patients were admitted from the causality and out patient department.

All the cases were selected with the following criteria:

- **Inclusion criteria:**
  1. Singleton pregnancy.
  2. Presence of live fetus.
  3. Period of gestation 37 - 42 weeks by date.

- **Exclusion criteria:**
  1. Intrauterine fetal death.
2. Multiple pregnancy.
3. The enrolled patients were divided into two groups

**Study Group** - Term pregnancies which were clinically and / or sonologically diagnosed as case of intrauterine growth restriction (IUGR).

**Control group** - Normal uncomplicated singleton term pregnancies which were appropriate for gestational age.

- **STUDY DESIGN** - Prospective clinicopathological study.
- **STUDY GROUP** - 78 cases of study group with clinically and / or sonologically diagnosed IUGR were reviewed and compared with 84 normal control cases, delivered in labour room and / or operation theater of Dr Susheela Tiwari Government Hospital and Government medical college, Haldwani between July 2012 to June 2014.
- Detailed information was collected regarding the maternal characteristics:
  b). Socioeconomic status.
  c). Parity.
  d). Gestational age.
  e). Mean blood pressure.
  f). Other Medical disorders associated with pregnancy.
  • Newborn data includes

  Gestational age of delivery
  Mode of delivery
  Birth weight
  Neonatal outcome
  • The placenta and umbilical cord obtained from these patients were collected and preserved in formalin solution and sent to pathology department of Government medical college, Haldwani where histopathological examination was done by pathologist.

**Placenta**

a) **Gross findings**
1) Cord length
2) Weight after trimming the cord
3) Diameter of the placenta

b) **Histopathological findings**
1) Decidual vasculopathy
2) Villous infarct
3) Villous hypovascularity
4) Villous fibrosis
5) Villous edema
6) Perivillous fibrin

7) Intervillous thrombus
8) Chronic villitis
9) Chronic chorioamnionitis.
10) Calcification.

**In umbilical cord**

**Gross cord abnormality**
1) true knots
2) long cords,
3) nuchal/body cords,
4) abnormal cord insertion,
5) hypercoiled cords,
6) narrow cords with diminished Wharton's jelly

**Histologic changes:**
1) fetal vascular ectasia,
2) fetal vascular thrombosis,
3) and fetal thrombotic vasculopathy/avascular villi.

Based on the above mentioned characteristics detailed study was done to study pathologic findings of the placenta and umbilical cord with IUGR babies and the findings were correlated with the clinical findings and pregnancy outcome.

**Statistical analysis**

All statistical analyses were performed with the SPSS 18.0 program for Windows Student’s unpaired T was used for statistical evaluation of continues variable(age, weight, gestational period, and pathological findings).

Whereas for parity and maternal risk factors fisher’s exact test was used. For religion, booking status, mode of delivery and clinical findings chi square test was used. A p-value <0.05 was accepted to be statistically significant and p >0.05 was accepted to be statistically insignificant. Data dependent upon verbal explanations were depicted as frequency and %, data dependent upon laboratory parameters were depicted as mean ±SD.

### III. OBSERVATIONS AND RESULTS

**Demographic Profile:** There was no difference in the demographic profile of both the groups. The mean age of patients in control group was 26.37 years while it was 26.32 years in study group (p value = 0.82). There was also no statistical difference between the gestational age of both the group (the control group was 38.55 weeks compared to 37.86 IUGR cases) (p value =0.64).

Parity in both the groups was also comparable. In Control and Study group the majority of the women was multigravida (58.33% Vs 64.10%), but the relation is statically not significant (p value =0.36). Significantly larger percentage of patients who belonged IUGR group(35.90%) were unbooked
pregnancies as compared to control group (10.75%, p=0.001), which is clinically significant.

PLACENTAL FINDINGS

GROSS FEATURES – The Average placental weight (in gms) in control group is 444.49 gms and study group is 355.69 gms which is significant (p value = 0.001). The average Placental size size is 348.48 cm² in control group and 296.65 cm² which is significant (p value = 0.002).

Other Morphological features Average Placental ratio (ratio of placental weight to newborn weight) 0.14 Vs 0.15 (p value =0.912), presence of accessory lobe(s) 1.19% Vs 5.13% (p value=0.574) and presence of retroplacental hematoma 4.76% Vs 6.41% (p value = 0.891) were comparable and statically not significant.

HISTOLOGICAL FEATURES–
Decidual vasculopathy [5(6.41%)] Vs 26 (33.33%)] p value = 0.002 , Villous infarct [10 (11.90%) Vs 30 (38.46%)] p value = 0.001 , Increased ST knots [19 (22.62%) Vs 51 (65.38%)] p value =0.002 , Villous fibrosis [8 (9.52%) Vs 30 (38.46%)] p value = 0.006 , Chronic Chorioamnionitis [11 (13.10%) Vs 26 (33.33%)] p value 0.003 , Villous hypovascularity [6 (7.14%) Vs 21 (26.92%)] p value = 0.001 , Intervillous thrombus [7 (8.33%) Vs 20 (25.64%)] p value = 0.006 were statically significant whereas Chronic villitis [(13 (15.48%) Vs 14 (17.95%))P value = 0.867 , calcification [51 (65.38%) Vs 49 (58.33%)] p value = 0.457 and villous edema [9 (10.71%) Vs 12 (15.38%)] p value = 0.776 were not significant.

UMBILICAL FINDINGS

GROSS FEATURES –Type of cord insertion - The most common type of cord insertion in our study was central in 61(71.43 %), centrifugal Insertion 17(20.24%), marginal in 4 (4.76%) and velamentous in 2 (3.57%) in study group. It was central in 59 (75.64) centrifugal Insertion in 10 (12.82%), marginal in 4 (5.13%) and velamentous in 5(6.41%) in control group. There was no significant difference in the two groups (p value = 0.63).

The mean cord length is 62.20 ± 8.97 cms in the control group while 51.58 ± 8.30 cms in study group (p value < 0.001) which is significant among the other gross features Reduced Wharton’s jelly {12 (15.38%) Vs 1 (1.19%)} p value = 0.006 , True knot {7 (8.98%) Vs 1 (1.19%)} p value = 0.006 and cord around the neck were significantly associated with IUGR group (32.05% Vs (13.10%)) p value = 0.001 were significant where as presence of single umbilical artery is not significant.

HISTOPATHOLOGICAL FEATURES- In histopathological feature there is significantly larger percentage of cases 26(33.33%) who belonged IUGR group have fetal thrombosis vasculopathy that control group 07(8.33%). (p value = 0.002)

NEWBORN PROFILE-
Although the proportion of gross congenital anomaly presence is more in the Study group babies (9%) as compared to that in the Control group (2.4%) but the difference is not found to be statistically (p value = 0.09). These congenital anomaly includes anencephaly, hydrocephalous, omphalocele or gastroscysis, meningomyelocele. Average weight of babies born to control group is 3.2 kgs whereas in study group is 2.1 kgs which is significant. (p value = 0.002). Significant number 13 (16.7%) of IUGR babies were admitted in NICU compare to Control group 2 (2.4%) (p value = 0.002).

The percentage of male babies was more in the Study group (55.13%) as compared to that in the Control group (47.62%) but statically it is not significant. (0.782)

MATERNAL RISK FACTORS

Among the various risk factors, Pregnancy induced hypertension was significantly more 25 (32.05%) in study group than in control group 8 (9.52%). As per smoking is concerned , it is certainly higher in study group 4 (5.13%) than in control group 1 (1.19%) but the association is statically not significant. Prevalance of anemia in study group 27(34.62%) and control group 31 (36.90%) is not significant. The proportion of chronic diseases , which includes diabetes (including both type -2 as well as gestational), tuberculosis and heart disease was more 7 (8.97%) in study group than control group 4 (4.76%) but was not statically significant.

IV. CONCLUSION

- PIH is a significant risk factor for IUGR The incidence of IUGR is significantly high in unbooked antenatal cases. It is associated with increased number of operative deliveries and fetal morbidity and mortality.
- The study reveals that the placenta is smaller in size and weight in IUGR cases.
- Histopathological findings reveal that IUGR cases are associated with Decidual vasculopathy, Villous infarct, Increased ST
knots, Villous fibrosis, Perivillous fibrin and Chronic villitis.

• The mean cord length is less, reduced Wharton’s jelly, true knot and cord around the neck were significantly associated with IUGR group.

• In histopathological feature of umbilical cord which issignificantly belonged to IUGR group is fetal thrombosis vasculopathy.

• Placenta being the reservoir and transferring organ of the nutrition required for normal fetal growth, if it is affected due to hypertension or any other factor, it shows the histological changes which affect its function, thereby giving rise to intrauterine growth restriction in the fetus.

BIBLIOGRAPHY


