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Research Article

Histopathological evaluation of placenta in low-birth-weight babies at a tertiary care center of Nepal

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ABSTRACT

Background & Objectives: Pregnancy-related death and morbidity rates are significantly impacted by the prenatal care provided to expectant mothers. Low birth weight (LBW) is the single most important factor among the several causes of perinatal mortality; consequently, placentas from all LBW babies (LBWB) should be routinely investigated to determine the likely reason. The purpose of this study was to evaluate the pathological alterations in the placenta associated with LBWB.

Materials and Methods: This is a cross-sectional observational study performed at Dhulikhel Hospital, Kathmandu University School of Medical Sciences, Kavre, Nepal. In this study, 100 placentae were included. 44 placentae from full-term delivered babies and 56 from pre-term delivered babies, all of them weighing less than 2500 gms. A gross and microscopic examination of the placentae was done.

Results: 100 placentas from low-birth-weight babies were studied. Most of the mothers were in the age group of 26-30 years with primi contributing to 59%. Gross findings like infarction and calcification were noted in 8% and 29 % of the cases. Microscopic findings like Intervillous hemorrhage (44%), Syncytial knots (69%), Stromal fibrosis (24%), Basement membrane thickening (26%), Cytotrophoblastic hyperplasia (28%), Increased villous vascularity (12%), Fibrinoid necrosis (17%), Calcification (38%), Villitis (12%) and Chorangiosis (15%) were noted.

Conclusion: Histopathological study together with clinical examination of placenta is simple and cost-effective technique in identifying the features present in LBWB placentae.

Keywords: Calcification, Chorangiosis, Fibrinoid necrosis, Low birth weight, Placenta, Syncytial knots

INTRODUCTION

Low Birth Weight (LBW) can be caused by preterm birth or by intrauterine growth restriction (IUGR) [1]. In developing countries from Asia, LBW is largely attributed to intrauterine growth retardation as compared to prematurity in developed and African countries [2]. Factors related to mothers that lead to LBW include anaemia, lack of nutrition, genetic factors, congenital or acquired heart conditions, respiratory diseases, kidney disorders, drug addiction, smoking, alcoholism, medications, diethylstilbestrol and anti-cancer drugs.

Factors related to the fetus that led to low birth weight include genetic influences, chronic infections, chromosomal abnormalities and multiple pregnancies. Placental factors contrubuting to Low Birth weight are abruptio placenta, placenta previa, thrombosis, infarction, deciduitis, vasculitis, chorioamnionitis, placental cyst and chorioangioma. All the Contributing factors are son interconnected that it is impossible to single out any one factor as solely causative [3].

Fetal development is primarily influenced by the nutrient supply to the fetus. The fetus is positioned at the end of a supply chain that facilitates the transfer of nutrients from the maternal/uterine circulation to the fetus via the placenta. The function of the placenta is crucial for the transfer of nutrients and

The fetus is positioned at the end of a supply c hain that facilitates the transfer of nutrients fr om the maternal/uterine circulation to the fet us via the placenta. The function of the placen ta is crucial for the transfer of nutrients and metabolites

metabolites between the mother and fetus [4].

Placenta is a crucial structure for fetomaternal circulation and there is direct relation between fetomaternal circulation and growth and development of the fetus. Placenta consists of lacuna, floating and anchoring villi, cytotrophoblast and syncytiotrophoblast, uterine blood vessels and uterine connective tissue [5]. In a histopathological evaluation of placenta in Intrauterine Growth Restriction (IUGR) pregnancies, the weight of IUGR placenta is less while infarction and intervillous fibrinoid deposition are high in IUGR placenta than normal placenta. In addition, among IUGR placenta, thickening of basal membrane and cytotrophoblast hyperplasia are more common. All the histopathological findings suggest placental and fetal blood flow reduction [6]. Significant numbers of syncytial knots, stromal fibrosis, and the number of capillaries in terminal villi are seen in the placenta of hypertensive and diabetic mothers [7].

Despite observed link between placenta and newborn health, histopathological examination of placenta is seldom performed in our institution and thus the etiology for LBW in such infants are not well defined. As there is a clear relationship between placental pathology and low birth weight of fetus, a thorough study of placenta is indispensible to evaluate possible etiological factors. Placental pathological examination is a reasonably simple technique and can be performed in our institution. Thus, this study can be helpful in finding the pathogenic mechanisms during pregnancies resulting in

low-birth-weight babies and in planning and management of future pregnancies.

MATERIALS AND METHODS

This cross-sectional observational study was done for one year from November 2023 to November 2024. All the biopsies labelled as Placenta received from all low birth weight newborns (<2500 gms) with singleton pregnancy in the Department of Pathology, Dhulikhel Hospital - Kathmandu University Hospital were included in the study. The ethical approval was obtained from institutional review board of Dhulikhel hospital, Kathmandu University (Ref No. 227/23) approved on 30th November 2023

Parenchyma was examined for calcification and infarction.

Cord - was cut at least 5 cm away from its site of attachment and was examined for:

- Cut sections for the patency of vessels.
- Thrombosis or hemorrhage
- Insertion of cord
- Surface discoloration, plaque
- Cyst/tumor
-

The slides were examined for following pathological findings:

- Intervillous hemorrhage
- Syncytial knots
- Stromal fibrosis
- Basement membrane thickening
- Cytotrophoblastic hyperplasia
- Increased villous vascularity
- Fibrinoid necrosis
- Calcification
- Villitis
- Chorangiosis

Placentae from all low-birth-weight newborns with singleton pregnancy were included in our study. Placenta from multiple pregnancy, intrauterine fetal death and neonatal congenital malformations were not included in our study. The specimens are stored in the grossing room of the department of Pathology. Normal cases will be discarded after six months while the specimens with important findings will be stored for a longer duration of time. Data analysis was done using Statistical Package for Social Sciences (SPSS 20.0) (SPSS Inc., Chicago, IL, USA).

RESULTS

In our study, 100 placentas of preterm as well as full term low birthweight babies with weight <2.5kg were studied. Age group of mother's ranges from 17-42 years, most of them are between 26-30 years of age with mean age being 27.99 years (Table 1).

Table 1: Age distribution

Age group	No. of cases
<20	3
20-25	25
26-30	45
31-35	14
>35	13
Total	100

Table 2 depicts that the primi-mothers contributed to 59 cases (59%) and multipara contributed to 41 cases (41%).

Table 2: Parity of patients

Parity	No. of cases
Primi	59
Multi	41
Total	100

All the placentae in the study had eccentrically attached cord. 58 of the placentae were between 300-400 gms in weight. Average weight of the placenta in the study was 323 gm. (Table 3)

Table 3: **Weight of placenta**

Placenta weight	No. of cases
<300 gms	12
300-400 gms	58
>400 gms	30
Total	100

Other gross findings like infarction and calcification are mentioned in the table 4.

Table 4: Percentage of cases displaying infarction and calcification

Gross findings	No. of cases
Infarction	8
Calcification	29
None	63
Total	100

Most of the new born are in the group of weighing between 2.1-2.49kg (71 cases), average weight being 2335 gms. Microscopic findings in our study are mentioned below in the Table 5.

Table 5: Microscopic examination findings

Microscopic findings	No. of cases
Intervillous hemorrhage	44
Syncytial knots	69
Stromal fibrosis	24
Basement membrane thickening	26
Cytotrophoblastic hyperplasia	28
Increased villous vascularity	12
Fibrinoid necrosis	17
Calcification	38
Villitis	12
Chorangiosis	15
Total	100

DISCUSSION

The placenta is crucial for proper fetal development as it supplies nutrients to the fetus. Intrauterine growth restriction may arise from maternal, fetal, placental or unidentified factors; however, the fundamental pathophysiology stems from either decreased nutrient availability in the mother or diminished transfervia the placenta to the fetus, and it may also relate to the fetus's lower utilization [8]. Other maternal influences linked to low birth weight include preterm birth, previous low birth weight, maternal age, height, hemoglobin level, iron supplementation, and the number of antenatal care visits [9].

A total of 100 placentae from babies weighing <2.5 kg was studied. The placenta of babies weighing 2.1-2.49 kg was maximum in number, followed by 1.6-2.0 kg. In our study maximum number of placenta were obtained from age group of 26-30 years mothers. In the study done by Jadhav CR et. al., 50 low birth placentae were studied in which the babies falling in weight group 1.6-2.0 kg were maximum and the maximum number of placenta were obtained from age group of 21-25 years mothers [8]. In our study, most of the placentae were obtained from male babies (55%) and female babies (45%). Similar results were found in the study done by Sharma AK et. al. i.e. male babies (60%) and female babies (40%) [2].

Among them 54 babies were delivered vaginally, followed by 46 cases delivered via lower segment caesarean section LSCS. Sharma AK et. al. found vaginal delivery in 35 cases while LSCS was done in 24 cases out of total 59 cases [2]. In the present study, placenta weighing 300-400 gms constituted

58% of all cases. In the study done by Sajid E et. al., 37% of the cases, the placenta weighed <400 gms [10]. Microscopically in our study, there was intervillous fibrin/hemorrhage (44%) which was similar to study done by Jadhav CR et al which was 56% and whereas it was 49% in study done by Sajid E et al [8-10]. In stromal fibrosis, increased villous fibrosis is associated with LBWB which can lead to reduction in the functioning of the villi causing placental insufficiency [8]. Stromal fibrosis was seen in 24% of the cases in our study. In conrast, in the study by Jadhav CR et al., stromal fibrosis was seen in 48% of the cases [8].

Increased syncytical knot signifies excessive proliferation of chorionic villous capillaries caused by diminished blood flow from various maternal or fetal conditions ultimately resulting in a low- birth weight babies at term [11]. In our study, it was seen in 69% similar to Kotgirwar et al (60%) whereas it was only 45% in the study done by Jadhav CR et al [8-12]. Macroscopically found infarction of the placenta is seen as necrosis microscopically. While placental infarction is prevalent in the later stages of pregnancy, numerous instances have been reported in different stages of gestation [13]. In our study, infarction was present in 8% of LBW cases which was statistically not significant. The percentage of infarction present in the study done by Mardi K. et al. and Acharya V. et al. were 28% and 58% respectively which was statistically significant while Kotgirwar et al. observed placental infarction in 1.8% cases which was not statistically significant similar to our study [12,14,15]. In our study, intervillous fibrin deposition was seen in 44% cases. In the study done by Mardi et al., it was 64% [14].

CONCLUSION

Morphological examination of the placenta is easy to perform, does not require an elaborate set up and can be performed routinely in a histopathological laboratory. It was found that, placental findings in our study ultimately point towards reduced uteroplacental blood flow and chronic uteroplacental insufficiency. On gross examination, we found that mean placental weights were on lower side in LBW newborns. Microscopic findings like intervillous fibrin deposition, stromal fibrosis, fibrinoid necrosis, calcification, syncytial knots strongly suggest uteroplacental insufficiency in our study.

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