LOW BIRTH WEIGHT LIVE BIRTHS: AN INSIGHT INTO PLACENTAL PATHOLOGYSurya Babu Sunkesula¹, Lingeswara Rao B², Tamil Arasi D. S³**HOW TO CITE THIS ARTICLE:**

Surya Babu Sunkesula, Lingeswara Rao B, Tamil Arasi D. S. "Low Birth Weight Live Births: An Insight into Placental Pathology". Journal of Evolution of Medical and Dental Sciences 2015; Vol. 4, Issue 16, February 23; Page: 2645-2651, DOI: 10.14260/jemds/2015/382

ABSTRACT: BACKGROUND AND PURPOSE: To study various pathological changes that occurs in the placentas of low birth weight live births and to obtain probable etiological causes. **MATERIALS AND METHODS:** Fifty placentas of low birth weight (LBW) live births collected were studied evaluating both gross and microscopic features. **RESULTS:** Anemia and PIH are the most common conditions that produce LBW live births. Placental morphological changes increase in direct proportion to the severity of the disease. Membrane opacities (48%), perivillous fibrin (46%) and placental infarcts are the most commonly observed morphological changes in LBW live births. Syncytial knots (88%) and fibrinoid necrosis (88%) are the most common histological findings in severe anemia. **CONCLUSION:** Study of the placenta will help to understand the role of the placenta in LBW live births and may give an insight into the etiopathogenesis of intrauterine growth retardation.

KEYWORDS: LBW live births, placenta, anemia, PIH.

INTRODUCTION: Placenta is the only organ to develop in adulthood and is the only organ with a defined end state. The placenta in the preserved state can relate foetal outcome with its histomorphology.

Even now, in an era of advanced medical care, the incidence of low birth weight and birth defects in the newborn is a major health problem in our country. This also may cause great financial stress to the parents as well as health care system, due to prolonged treatment in NICU.

In general, neonates weighing less than 2.5 kg at birth are termed as low birth weight (Both preterm and term). In both groups, pathological examination of the placenta may reveal the causes of low birth weight, and gives useful insight into the diagnosis and treatment of sick newborns and reflects the impact of maternal disorders of pregnancy.

This study focuses on the morphological appearance of the placenta in relation to the low birth weight live births, and the pathological changes in the various placentas collected from high risk mothers who had delivered low birth weight live babies.

MATERIALS AND METHODS: This is a one year prospective study from January 2012 to December 2012 conducted in our hospital. Fifty placentas of live births with birth weight of less than 2.5 kg, expelled during normal delivery or caesarian section were studied. Placentas of abortion, intrauterine death and of still born were excluded.

Placentas were examined in fresh state after delivery, handling the specimens with great care avoiding lacerations. Examination of the membranes, umbilical cord, fetal and maternal surfaces of the placenta was done, sections were taken from the respective areas, fixed for 24 hours and H & E slides were prepared for microscopic examination.

ORIGINAL ARTICLE

OBSERVATIONS AND RESULTS: Out of the total 50 placentas from low birth weight live births, 38 were from high risk pregnancies and 12 were from uneventful pregnancies. Of them, 24 were term deliveries and 26 were preterm deliveries.

High risk factors	Total no. of placentas	Percentage
Anemia	12	24%
PIH	11	22%
Gestational DM	03	6%
Abruptio placenta	01	2%
Rh negative	02	4%
Heart disease	01	2%
Cervical incompetence	01	2%
Twin gestation	01	2%
Oligohydramnios	06	12%
Uneventful	12	24%
Total	50	100%

Table 1: Shows various risk factors of pregnancy in the present study

Of the 50 low birth weight live births, 25 babies had weight range from 1500g to 2499g (low birth weight), 24 were between 1000-1499g (very low birth weight) and one baby had birth weight of 950g (extremely low birth weight).

Placental weight ranged from 200-500g.

Table 2: The gross morphological changes observed in placentas are represented

Gross morphological changes	Total number	Percentage
Placental infarct	14	28%
Perivillous fibrin	23	46%
Membrane opacities	24	48%
Calcifications	10	20%
Umbilical cord false knots	03	6%
Single umbilical artery	03	6%
Subchorial haematoma	02	4%
Retroplacental haematoma	01	2%
Circumvallate placenta	01	2%
Accessory lobe	01	2%

Fig. 1: Showing (A) placenta with peripheral incision of umbilical cord (B) abruptio placenta

Table 3: The histopathological changes in placentas are shown

Histological changes	Total number	Percentage
Perivillous fibrin	23	46%
Infarct	14	28%
Calcifications	10	20%
Chorioamnionitis	24	48%
Increased syncytial knots	44	88%
Fibrinoid necrosis	44	88%
Increased villous cytotrophoblast	33	66%
Basement membrane thickening	25	50%
Deficiency of vasculosyncytial membrane	17	34%
Stromal fibrosis	16	32%
Villous hypovascularity	18	36%
Intravillous hemorrhage	25	50%
Distal villous hypoplasia	6	12%

Fig. 2: Showing (A) increased syncytial knots (B) calcifications, (C) fibrinoid necrosis (D) distal villous hypoplasia

The most commonly observed changes were increased syncytial knots (44 cases) and fibrinoid necrosis (44 cases). Chorioamnionitis was observed in 24 placentas and perivillous fibrin deposit in 23 placentas. Other common findings were villous hypervascularity, increased villous cytotrophoblast, deficiency of vasculo-syncytial membranes, basement membrane thickening, intervillous hemorrhage and calcification. In anemia and PIH, the degree of these changes was proportionate to the severity of the disease.

DISCUSSION: The placenta has been described as the mirror of the perinatal mortality. Intrauterine growth retardation is a complication of many pregnancies. The factors responsible for growth retardation include maternal malnutrition, anaemia, preeclampsia, maternal infection, drug abuse, genetic factors and genetic diseases, congenital malformation, multiple gestations, placental/cord abnormalities and maternal smoking. Growth and survival of the fetus is essentially dependent on development, formation, maturation and function of placenta. Hence, a study of the morphology of

ORIGINAL ARTICLE

the placenta is very important and useful in learning its relationship and its predictive value to fetal weight and possible birth defects.

This study mainly deals with both gross and histopathological changes that occurred in low birth weight live births. The placental parameters like weight and size of the placenta were significantly less than normal in preterm and term low birth weight deliveries. Out of 50 placentas, 26 were from preterm deliveries. Among 26 preterm placentas, 18 were weighing less than 300g. this finding is comparable with Virupaxi et al.^[1] In high risk pregnancies like anemia and PIH, the placental weight is significantly reduced with the severity of the disease.^[2]

In this study, the average mean weight of placenta in low birth weight live births was 300g. Nobis and Das in their study have shown that the placental weight in toxemic cases varies from 279 to 407 grams.^[3]

In the present study, 40 placentas (80%) had normal discoid shape which had no significance in low birth weight placentas. One placenta had accessory lobe being 2% in the present study. Umbilical cord attachment is a specialized anatomy of the placenta and fetus.^[4] The commonest mode of insertion of the umbilical cord was peripheral in 56% of cases followed by central in 44% of cases. Velamentous, battle door, furcated insertion were not observed in the present study. In the study done by Virupaxi et al the umbilical cord insertion was more towards the margin with the increase in severity of anemia.^[1] In the present study, peripheral insertion of the umbilical cord was seen in 10 cases of anemia complicating pregnancy.

False knots were noted in 3 cases (6%) and thrombosis noted in 1 case (2%) in this study. False knot had no clinical significance but thrombosis of the cord was associated with severe preeclampsia.^[2]

In the present study, there were 3 single umbilical artery (SUA) placentas, two of which were associated with twin gestation and one with gestational diabetes mellitus. Many observations have been published about this umbilical cord maldevelopment which was seen in 7% twin gestations and 1% of term pregnancies.^[5,6]

The most common cause of chorioamnionitis in human beings is by ascending infection.^[7] The association between chorioamnionitis and fetal growth restraint is well established. In this study, grossly membrane opacities were seen in 24 placentas (48%). Corresponding histopathological changes were consistent with chorioamnionitis.

Placental infarction of >5% surface area is considered pathological and more frequently seen in toxemia and IUGR due to thrombotic occlusion of maternal uteroplacental vessels.^[8] Intrauterine hypoxia leads to coagulation necrosis of the villus tissue, secondary to the occlusion of the placental vessels in cases of improper vascular adaptation during placentation.^[9] Among 50 placentas, placental infarcts were observed in 14 cases (28%). This is almost comparable with the study done by Mirchandhini et al.^[10] Out of the 14 cases, 10 were from PIH. This correlates with the study done by Aparna Narasimha et al.^[2]

In general, calcification is regarded as placental senescence or degeneration.^[11] The incidence of calcification of the placenta in the present study was 20%. This was most commonly associated with preeclampsia and IUGR. This is almost agreeable with the study done by Virupaxi et al in which 22% of cases showed calcification which were associated with PIH.^[2]

The incidence of retroplacental hematoma was 2%. This case was associated with abruption placenta. Subchorionic thrombosis was seen in 2 placenta. One case of IUGR has been reported in association with massive subchorial thrombosis.^[8]

ORIGINAL ARTICLE

Syncytial knots are seen with increased frequency in the last weeks of pregnancy and more villi show these changes in high risk pregnancies.^[12,13] Out of 50 placentas, 44 showed increased number of syncytial knots (88%) in the present study.

Fibrinoid necrosis is nothing but a fibrinoid patch that replaces villous stroma and the vasculature underneath a more or less intact trophoblastic cover. It occurs occasionally in normal mature placentas, but the incidence is increased in complicated pregnancies. Significant villous fibrinoid necrosis was noted in 44 cases (88%) in the placentas of PIH, anemia and IUGR. These findings are in concordance with other studies.^[2,8]

The incidence of villi showing a thickened basement membrane in more than 3% of the villous population is regarded as abnormal and is a common feature of placentas from complicated pregnancies. In the present study, 25 placentas showed increased basement membrane thickness (50%). These findings concurred with those of other authors.^[14,15]

In the present study, 32% of placentas showed stromal fibrosis. There was an increased incidence of fibrotic placenta in pregnancy complicated by Fox et al, also correlated with this finding.^[14]

Vasculosyncytial membrane (VSM) is an index of fetal hypoxia. The incidence of VSM deficiency was noted in 34% of cases. The paucity of VSM was seen in higher grades of PIH, correlating with the severity of disease.

Chorangioma is an expansile nodular lesion composed of capillary vascular channels, intervening stromal cells and surrounding trophoblasts. Intermediate sized chorangioma are associated with IUGR.^[16] In the present study, one case of chorangioma was found.

SUMMARY: To summarize, placental morphological changes increase in direct proportion to the severity of diseases in high risk pregnancies. Study of these features in placenta will help to understand the role of the placenta in LBW live births and may give an insight into the etiopathogenesis of intrauterine growth retardation.

REFERENCES:

1. R.D. Virupaxi, B. R. Potturi, V. S. Shirol, Desai S. P, V. B. Hukkeri. Morphology of Placenta and its Relation with Small for Date Babies in 950 Live Births. Recent Research in Science and Technology. 2011; 3 (2): 123-126.
2. Narasimha A, Vasudeva D S. Spectrum of changes in placenta in toxemia of pregnancy. Indian J Pathol Microbiol 2011; 54: 15-20.
3. Nobis P, Das U. Placental morphology in hypertensive pregnancy. Journal of Obstetrics and Gynecology of India. 1991; 41: 166-169.
4. E. Cullen. Embryology, anatomy, and diseases of the umbilicus together with diseases of the urachus. Philadelphia, Saunders, and London, 1916.
5. HATHOUT H. THE VASCULAR PATTERN AND MODE OF INSERTION OF THE UMBILICAL CORD IN ABORTION MATERIAL. J Obstet Gynaecol Br Commonw. 1964 Dec; 71: 963-4.
6. Sepulveda W, Nicolaidis P, Bower S, Ridout DA, Fisk NM. Common iliac artery flow velocity waveforms in fetuses with a single umbilical artery: a longitudinal study. Br J Obstet Gynaecol. 1996 Jul; 103 (7): 660-3.
7. Driscoll SG. Antenatal infection and unruptured membranes. Pediatrics. 1970 May; 45 (5): 886.

ORIGINAL ARTICLE

8. Thomas D, Makhoul J, Müller C. Fetal growth retardation due to massive subchorionic thrombohematoma: report of two cases. J Ultrasound Med. 1992 May; 11 (5):245-7.
9. Kher AV, Zawar MP. Study of placental pathology in toxemia of pregnancy and its fetal implications. Indian J Pathol Microbiol. 1981 Oct; 24 (4): 245-51.
10. Mirchandani JJ, Mallik GB, Chitra S. Correlation of foetal outcome with some pathological changes of placenta. J Obstet Gynaecol India 1979; 29: 1131-9.
11. Shanklin, d.r; (1970): The influence of placental lesions on the newborn infant: pediatric clinics of north america 17, 25-42.
12. Mallik GB, Mirchandani JJ, Chitra S. Placenta in intrauterine growth retardation. J Obstet Gynaecol India 1979; 29:805-10.
13. Karsdorp VH, Dirks BK, van der Linden JC, van Vugt JM, Baak JP, van Geijn HP. Placenta morphology and absent or reversed end diastolic flow velocities in the umbilical artery: a clinical and morphometrical study. Placenta. 1996 Sep; 17 (7): 393-9.
14. Fox H. General pathology of the placenta. In: Fox H, editor. Haines and Taylor Obstetrical and Gynecological Pathology. 4th ed. Edinburgh: Churchill Livingstone: 1995. P. 1480-2.
15. Masodkar AR, Kalamkar LR, Patke PS. Histopathology of placenta and its correlation with fetal outcome. J Obstet Gynaecol India 1985; 35: 294-7.
16. Mucitelli DR, Charles EZ, Kraus FT. Chorioangiomas of intermediate size and intrauterine growth retardation. Pathol Res Pract. 1990 Aug; 186 (4): 455-8.

AUTHORS:

1. Surya Babu Sunkesula
2. Lingeswara Rao B.
3. Tamil Arasi D. S.

PARTICULARS OF CONTRIBUTORS:

1. Associate Professor, Department of Pathology, Kurnool Medical College, Kurnool, Andhra Pradesh.
2. Senior Resident, Department of Pathology, Kurnool Medical College, Kurnool, Andhra Pradesh.
3. Professor and HOD, Department of Pathology, Kurnool Medical College, Kurnool, Andhra Pradesh.

FINANCIAL OR OTHER

COMPETING INTERESTS: None

NAME ADDRESS EMAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Tamil Arasi D. S,
Professor and HOD,
Department of Pathology,
Kurnool Medical College,
Kurnool-518002, Andhra Pradesh.
E-mail: tamlarasid48@gmail.com

Date of Submission: 05/02/2015.
Date of Peer Review: 06/02/2015.
Date of Acceptance: 13/02/2015.
Date of Publishing: 20/02/2015.