ORIGINAL RESEARCH

Morhological study of placenta in low birth weight babies: A histopathological study

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Received: 12 March, 2023 Accepted: 18 April, 2023

ABSTRACT

Aim: The aim of the present study was to assess the spectrum of morphological features in placentae of low birth weight babies and compared the morphological findings with maternal factors/ fetal factors associated with LBW. **Methods:** The present prospective study was conducted in the Department of Pathology in Maharishi Markandeshwar Medical College and Hospital, Kumarhatti, Solan for period of 1.5 year, dates subject to IEC approval. Minimum 100 Cases of placentae of low birth weight (<2.5 kg) at >28 weeks of gestation and 100 cases of controls were included in the study. **Results:** The age of the mothers in the case group ranged from 17-41 years with maximum mothers (64) in the age group of 21-30 years. The age of the mothers in the control group ranged from 19 years to 42 years with maximum mothers (73) in the age group of 21-30 years. In the case group (n=100), 57 mothers were multigravida and 43 mothers were primigravida. In the control group (n=100), 51 mothers were primigravida and 49 mothers were multigravida. In the case group (n=100) 52 mothers had term deliveries and 48 mothers had preterm deliveries. In the control group (n=100) all the mothers had term deliveries. All mothers included in the control group had uncomplicated pregnancies. In the case group mothers had single or multiple maternal and fetal risk factors. The most common risk factor was preterm deliveries (48), followed by hypothyroidism (16), anemia (16), PIH (10), PROM (9), Rh-ve pregnancy (6), IUGR (6), preeclampsia (5), placenta previa (4), and GDM (4). **Conclusion:** The present study concluded that the placental pathologies are not single but rather multiple in low birth weight deliveries. The prevalence of placental pathology in low birth weight infants is rather high and the examination of placenta provides considerable amount of data in such cases.

Key words: Placenta, low birth weight, histopathology

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INTRODUCTION

Low Birth Weight (LBW)infants have high prevalence of placental pathology (80-90%).[1,2] UNICEF-WHO defined Low birth weight (LBW) as "birth weight less than 2500g (<5.5lbs) irrespective of the gestational age".[3] Very LBW is ≤1500g and extremely LBW is ≤1000g.[4] In India, National Family Health Survey-4 data (2015-16) documented 18% of babies with LBW for 78% of live births.[5] According to District Level Household and FacilitySurvey-4 in Himachal Pradesh incidence of LBW is 13.8%.[6] Fetal growth and the birth weight can be affected by maternal, fetal and placental factors. The placentae of LBW babies show various types of lesions. Placental lesions accumulating over adequate time interval may lead to LBW and further fetal growth retardation. Major histopathological changes in the low birth weight placenta point towards decreased blood flow to the placenta resulting in chronic placental insufficiency. Commonly observed histopathological lesions in placentae of LBW babies is placental infarction which can be secondary to compromise of uteroplacental circulation.[7,8] This causes reduction in transfer of oxygen and nutrients to the fetus which explains LBW.

Low birth weight lower than that expected from the genetic potential might be caused by fetal, maternal or placental factors or a combination of risk factors,

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resulting in an impaired placental transport of nutrients or reduced growth potential of the fetus. Constitutional, gender and hereditary factors explain up to 40% of the variability of birth weight. Maternal age (<20 or >35 yrs), ethnicity, marital status, birth interval, educational level and socio-economic conditions are other explanatory factors. Common fetal factors are genetic and/or chromosomal aberrations. Medical risk factors for LBW before pregnancy are chronic conditions like hypertension, renal insufficiency, cardio-respiratory, autoimmune, endocrine or infectious disorders. The risk factors for LBW during pregnancy are hypertensive disorders, diabetes, malnutrition, bleeding, anemia, infection, placental or fetal anomalies and multiple pregnancies. The morbidities of term and moderately preterm (>32 weeks) LBW are mainly related to uteroplacental insufficiency and poor energy substrate transfer, resulting in neonatal complications like birth asphyxia, hypothermia, meconium aspiration, polycythaemia, hypoglycemia, hypocalcaemia and thrombocythaemia.[9] Histopathological examination of placenta gives a better understanding of the pathogenesis and causes of LBW. It can further help in management of future pregnancies. All placentae of LBW infants should be examined. There is a high incidence of LBW in Himachal Pradesh and only a limited number of studies have been conducted in this region. In this study we will look for placental pathology and correlate maternal and fetal factors with placental pathology in LBW babies.

The aim of the present study was to assess the spectrum of morphological features in placentae of low birth weight babies and compared the morphological findings with maternal factors/ fetal factors associated with LBW.

MATERIALS AND METHODS

The present prospective study was conducted in the Department of Pathology in Maharishi Markandeshwar Medical College and Hospital, Kumarhatti, Solan for period of 1.5 year, dates subject to IEC approval. Minimum 100 Cases of placentae of low birth weight (<2.5 kg) at >28 weeks of gestation and 100 cases of controls were included in the study. Controls was placentae of babies with weight more than 2.5 kgs at >28 weeks of gestation. Mothers with Multiple pregnancies (Twins/Triplets and higher order), Intrauterine death (IUD) and Still birth were excluded.

Placentae from the mothers delivering low birth weight babies >28 weeks of gestation, were received from Obstetrics and Gynecology Department immediately after the delivery both from normal deliveries and Caesarean sections. The placenta was placed in wide mouth jar containing 10% formalin and was delivered to histopathology laboratory. In each case, a preliminary history, Antenatal clinic records, radiological investigations and other relevant laboratory investigations was recorded.

Any visible anomalies, sex, weight, and APGAR score of the baby at the time of birth werel also be recorded. The collected placenta was examined for following parameters:

Placental disc-

1. Weight.
2. Shape.
3. Dimensions.
4. Number of lobes.
5. Number of Cotyledons
6. Membranes-color/opacity.

Umbilical cord-

1. Length of cord.
2. Insertion.
3. Hematoma.
4. Cord knot.
5. No. of vessels.

After gross examination longitudinal cuts were made through the maternal surface at a distance of 1-2 cm in a bread loaf manner. Then it was kept for fixation in 10% formalin for 24-48 hrs. The placentae was to be grossed and at least 5 sections was taken: two sections from center of placenta, one section from periphery of placenta, one transverse section of umbilical cord and one free membrane bit from a membrane roll. Apart from these, grossly abnormal areas like infarct and calcified areas were also sampled. Hematoxylin and Eosin staining. Masson Trichome and PAS stain were used wherever required.

THE TISSUE SECTIONS WERE THEN BE STUDIED IN DETAIL BY LIGHT MICROSCOPY. FOLLOWING PARAMETERS WERE STUDIED

1. Placental calcification
2. Infarct.
3. Peri villous/intervillous fibrin deposit.
4. Inflammation of membranes, villi and umbilical cord.
5. Maternal and fetal vasculature.
6. Syncytial knots.

STATISTICAL ANALYSIS

Descriptive statistics were used to analyze the data. Chi square test was used to compare categorical variables and Student t test was used to compare continuous variables in the two groups (cases and controls).

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RESULTS

Table 1: Demographic details

Variables	Case	Controls
Age groups		
21-30 years	64	73
31-40 years	26	20
>40 years	10	7
Parity		
Primiparous	43	51
Multiparous	57	49
Term deliveries		
Preterm	48	-
Term	52	100
Mode of delivery		
NVD	54	56
LSCS	46	44

The age of the mothers in the case group ranged from 17-41 years with maximum mothers (64) in the age group of 21-30 years. The mean age in the case group was 28.3 ± 5.3 years. The age of the mothers in the control group ranged from 19 years to 42 years with maximum mothers (73) in the age group of 21-30 years. The mean age in control group was 27.5 ± 4.6 years. In the case group (n=100), 57 mothers were multigravida and 43 mothers were primigravida. In the control group (n=100), 51 mothers were primigravida and 49 mothers were multigravida. In the case group (n=100) 52 mothers had term deliveries and 48 mothers had preterm deliveries. In the control group (n=100) all the mothers had term deliveries. In the case group (n=100), 54 mothers had Normal Vaginal Delivery (NVD) and 46 mothers had Lower segment caesarean section (LSCS). In the control group (n=100), 56 mothers had NVD and 44 mothers has LSCS.

Table 2: Birth weight of babies in case and controls group

Birth weight	N%
Cases	
1501- 2499 gm (LBW)	88 (88)
1001- 1500 gm (VLBW)	9 (9)
≤ 1000 gm (ELBW)	3 (3)
Controls	
2500-3000 gm	69 (69)
3001-3500 gm	29 (29)
3501-4000 gm	2 (2)

Table 3: Gross Findings of placentae

Gross findings	Case	Controls	P Value
Placental weight (gm)			
Range	200-550	350-750	
Mean ± SD	423 ± 89	540 ± 86	<0.001
Placental Maximum diameter (cm)			
Range	10-20	14-24	
Mean ± SD	15.8 ± 1.9	17.4 ± 1.7	<0.001
Placental thickness (cm)			
Range	1-3.5	1-4	
Mean ± SD	2.1 ± 0.6	2.4 ± 0.7	<0.001
Cotyledons (numbers)			
Range	4-14	5-17	
Cord length (cm)			
Range	0.7-46	7-49	
Mean ± SD	19.9 ± 8.05	20.3 ± 8.3	<0.001

The gross findings of placenta were found to statistically significant.

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Table 4: Microscopic findings

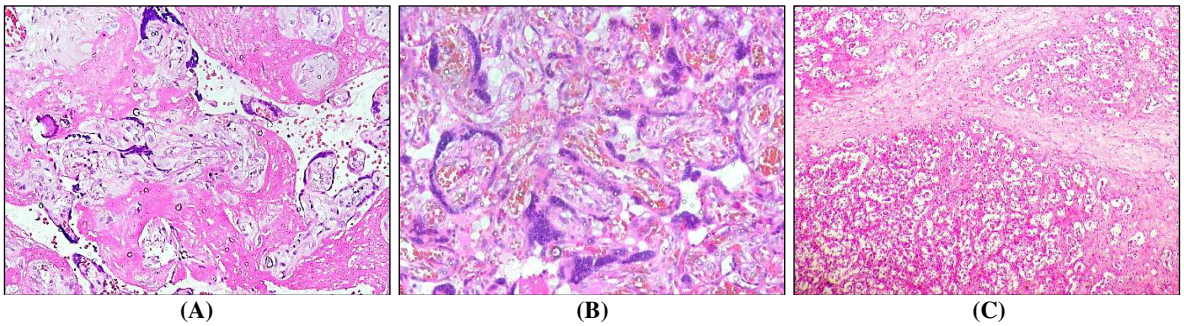
	Cases	Controls	
Microscopic findings	**No. of placentae**	**No. of placentae**	**p-value**
Increased syncytial knots	65	14	<.001
Calcification	50	30	.003
Chorangiosis	47	18	<.001
Infarct	43	18	<.001
Stromal fibrosis	30	12	.001
Medial hypertrophy with septations	28	0	-
Perivillous fibrin deposition	22	11	.036
Chorioamnionitis	19	3	<.001
Chorangioma	1	0	-

The microscopic findings were found to be statically significant.

Table 5: Microscopic findings in relation to maternal/fetal factors

Cases	Calcification	Infarction	Perivillous /Intervillous fibrin deposition	Stromal fibrosis	Increased Syncytial knots	Chorioamnionitis	Chorangiosis	Medial hypertrophy and septation
Preterm deliveries (48)	26 (54.1%)	23 (47.9%)	11 (22.9%)	17 (35 %)	30 (62.5%)	14 (29.1%)	17 (35 %)	17 (35 %)
Hypothyroidism (16)	8 (50%)	6 (37.5%)	2 (12.5%)	5 (31.2%)	9 (56.2%)	4 (25%)	12 (75%)	3 (18.7%)
Anemia (16)	9 (56.2%)	3 (18.7%)	2 (12.5%)	5 (31.2%)	14 (87.5%)	4 (25%)	8 (50%)	5 (31.2%)
PIH (10)	7 (70%)	5 (50%)	1 (10%)	3 (30%)	6 (60%)	1 (10%)	2 (20%)	3 (30%)
PROM (9)	3 (33.3%)	4 (44.4%)	1 (11.1%)	1 (11.1%)	5 (55.5%)	6 (66.6%)	5 (55.5%)	2 (22.2%)
Rh-ve pregnancy (6)	4 (66.6%)	2 (33.3%)	0	0	3 (50%)	1 (16.6%)	3 (50%)	1 (16.6%)
IUGR (6)	3 (50%)	3 (50%)	1 (16.6%)	3 (50%)	4 (66.6%)	1 (16.6%)	3 (50%)	1 (16.6%)
Preeclampsia (5)	3 (60%)	3 (60%)	0	0	4 (80%)	0	4 (80%)	1 (20%)
Placenta previa (4)	3 (75%)	2 (50%)	1 (25%)	2 (50%)	3 (75%)	0	0	2 (50%)
GDM (4)	3 (75%)	1 (25%)	1 (25%)	1 (25%)	3 (75%)	1 (25%)	3 (75%)	2 (50%)
Normal (17)	3 (17.6%)	5 (29.4%)	5 (29.4%)	5 (29.4%)	9 (52.9%)	0	10 (58.8%)	4 (23.5%)

All mothers included in the control group had uncomplicated pregnancies. In the case group mothers had single or multiple maternal and fetal risk factors. The most common risk factor was preterm deliveries (48), followed by hypothyroidism (16), anemia (16), PIH (10), PROM (9), Rh-ve pregnancy (6), IUGR (6), preeclampsia (5), placenta previa (4), and GDM (4).



(A) **(B)** **(C)**

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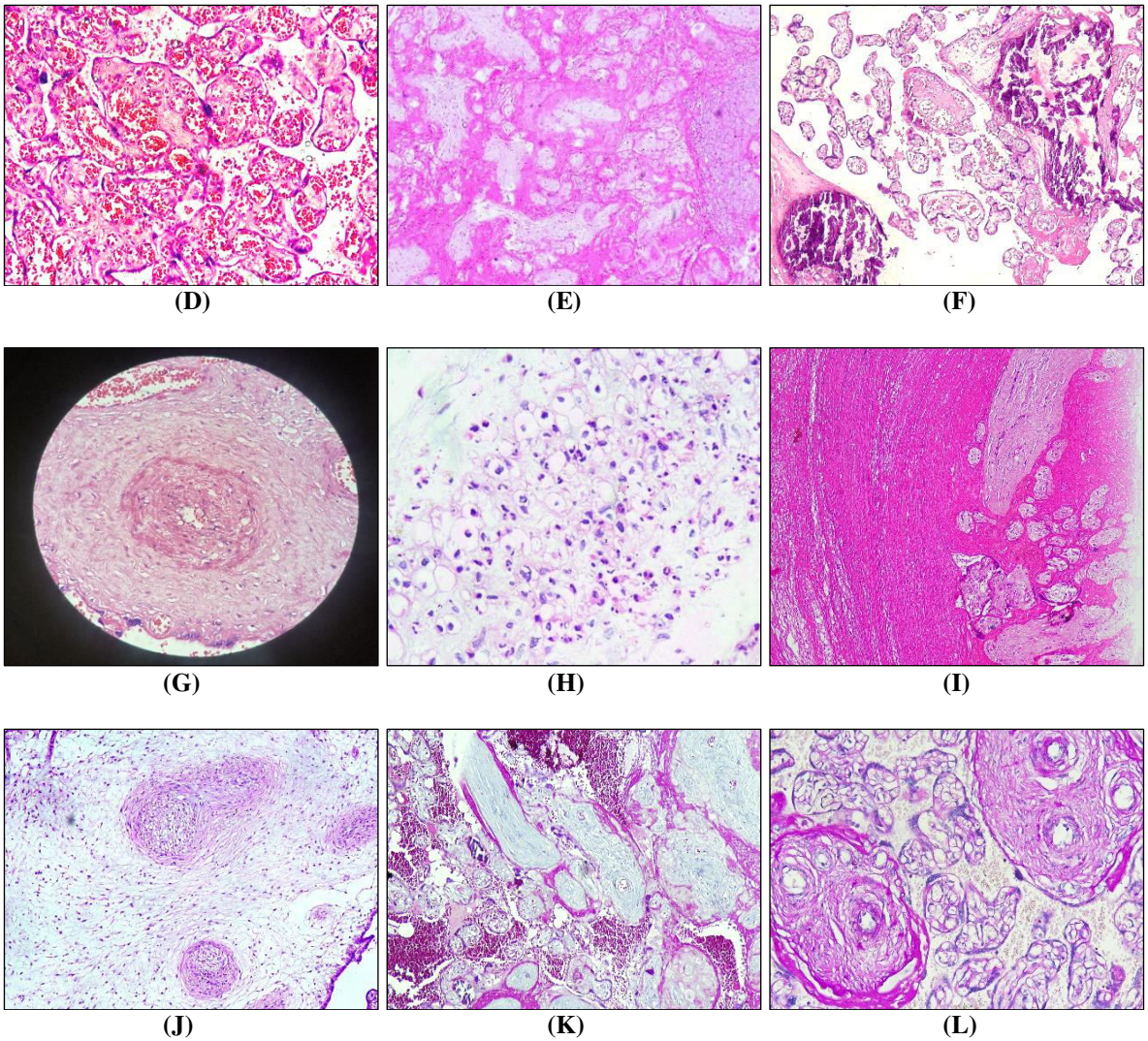

Figure A) Increased syncytial knots (H&E 400X), B) Perivillous fibrin deposition (H&E 400X) C) Chorangioma (H&E 400X), D) Chorangiosis (H&E 400X), E) Late Infarct (H&E 100X), F) Dystrophic Calcification (H&E 100 X), G) Medial Hypertrophy (H&E 400X), H) Chorioamnionitis (H&E 400X), I) Stromal fibrosis (H&E 100X), J) Thrombus (H&E 400X), K) Stromal fibrosis (Masson Trichome 100X) L) Basement membrane thickening (PAS 400X).

DISCUSSION

In the current study the age of the mothers in case group ranged from 17-41 years, where maximum mothers (64) were in the age group of 21-30 years (mean age 28.3 ± 5.3 years). The age of the mothers in control group ranged from 19 years to 42 years where maximum mothers (72) in the age group of 21-30 years (mean age 27.5 ± 4.6 years). The results were similar to Nigam *et al*., (2014)[10], Nkwabong *et al*., (2015).[9] In the present study, 51% mothers were primigravida and 49% were multigravida in the case group (n=100). While in control group 58% were multigravida and 42% were primigravida (n=100).

In the current study, the placental weight was in the range of 220-550 gms in case group. The mean weight was 423 ± 89 gm. In comparison the placental weight was in the range of 350-750 gm in the control group. The mean weight was 540 ± 86 gm, which was corresponding to Khajuria *et al*., (2019)[11] and Sanchita *et al*., (2022).[12] In the present study maximum placental diameter was in the range of 10-20 cm with mean diameter 15.8 ± 1.9 cm in the case group, whereas maximum placental diameter ranged from 14-24 cm with mean of 17.4 ± 1.7 cm in the control group. The results noted in the present study was nearly similar to the observations made by Sanchita *et al*., (2022)[12], Biswas *et al*., (2008)[13]and Kotigwar *et al*., (2011).[14] In our study placental thickness ranged between 1-3.5 cm with mean of 2.1 ± 0.6 cm in case group and in the control group the range of 1-4.5 cm with mean of 2.4 ± 0.7 cm. The results were similar to study conducted by Kotigwar *et al*., (2011).[14]

On gross examination 15% placentae showed infarct in case group (n=100) while 6% revealed infarct in control group (n=100) in the current study. Mardi *et al*., (2017)[15] (n=75) noticed infarct in 16% cases

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which was similar to our study. In the current study, cord insertion in both the groups was nearly similar to study conducted by Biswas *et al*., (2007)[13], Nigam *et al*., (2015).[10]

In the present study, 89% placentae in the case group (n=100) had shiny and intact membranes while 11% had meconium-stained membranes. In the control group (n=100), only 2% placentae had meconium-stained membranes. In a study conducted by Nigam *et al*., (2015)[10] on 60 cases, 33% of the placental membranes were meconium stained and none of the placenta was meconium-stained in the control group. In case group (n=100) the placental/fetal weight ratio was in the range of 0.12-0.5 with mean ± SD of 0.20 ± 0.05. In control group (n=100) the placental/fetal weight ratio was in range of 0.13-0.25 with mean ± SD of 0.18 ± 0.03. In the present study, in one of the cases placental coefficients was found to be 0.5, due to chorangioma in which fetal weight was 1000 gms and fetal weight was 550 gms. Lao *et al*., (1996)[16], incorporated 73 cases and 309 controls and reported placental to fetal ratio 0.19 and 0.18 respectively which was nearly similar to our study. Ruangvutilert *et al*., (2001)[17] studied 96 cases and 804 controls and reported placental to fetal ratio was 0.20 and 0.19 respectively which is almost similar to our study.

In the present study, increased syncytial knots were present in 65% of the placentae in the case group, the results of which were similar to study by Kotigwar *et al*., (2011)[14], Mardi *et al*., (2017).[15] The risk of LBW increases with the number of placental lesions. We reported increased syncytial knots in 14% of the placentae in the control group which is in concordance with studies by Romero *et al*., (2018)[18] and Mardi *et al*., (2017).[15] Calcification is a part of normal ageing and maturation process of placenta, but when it occurs prematurely it indicates pathological maturation and is associated with intrauterine growth retardation.18 In our study, 50% of the placentae in the case group and 30% in the control group showed calcification and results were in concordance with Nkwabong *et al*., (2015).[8] Chorangiosis was present in 47% of the placentae in the case group of our study and 18% of the placentae in control group. These observations were nearly similar to Sharma *et al*., (2021).[20] Increased percentage of chorangiosis in cases and control group in our study was probably due to mothers living in an areas with high altitude, with an average altitude of 1768.3m above the sea level. According to the pathways of villous angiogenesis due to villous oxygenation, uteroplacental hypoxia resulted in reduction of villous oxygen content and thereby producing greater amounts of highly vascularized terminal villi.[21]

In the present study, percentage of infarction in the case group was comparable to the study by Khajuria *et al*., (2019).[11] In the control group, infarction was present in 18% of the placentae in our study, which was concordant to study by Nkwabong *et al*., (2015)[8] and Nigam *et al*., (2014).[10] All mothers included in the control group had uncomplicated pregnancies. In the case group mothers had single or multiple maternal and fetal risk factors. The most common risk factor was preterm deliveries (48), followed by hypothyroidism (16), anemia (16), PIH (10), PROM (9), Rh-ve pregnancy (6), IUGR (6), preeclampsia (5), placenta previa (4) and GDM (4). Chronic uteroplacental insufficency is the dominating etiopathogenetic factor in preterm deliveries. In our study chorioamnionitis was similar to studies conducted by Ericksen *et al*., [22] and Stanek J *et al*., [23] Percentage of all other parameters was more in our study as compared to other studies. It was probably due to limited sample size in our study as compared to the studies conducted by other authors.

Hypothyroidism is associated with gross placental abnormalities that compromise placental function and hence fetal growth. In current study, calcification and increased syncytial knots were similar to study by Hudda S *et al*., (2022).[24] Anemia leading to hypoxia causes changes in placenta which increases the risk of fetal growth retardation and LBW. According to study conducted by Kaur *et al*., [25] reported increased syncytial knots in 78.6%, which corresponded with our study. Calcification was present in 56.2 % akin to studies by Kaur *et al*., [25] and Less percentage of perivillous fibrin deposition and infarction was noted in our study as shown in the table above. Pregnancy induced hypertension causes altered arrangement of intracotyledonous vasculature which leads to babies with low birth. Syncytial knots were less in our study as compared to other studies. Infarction and calcification was similar to study conducted by Mohan *et al*., [26] PROM is either caused by or can result in inflammation in the fetal compartment, insufficient maternal placental perfusion or both. PROM cases showed higher incidence of chorioamnionitis. The most common finding in our study was chorioamnionitis (66.6%). These findings were corresponding with Chellam *et al*., [27] who reported chorioamnionitis in 71.8%.

CONCLUSION

The present study concluded that the placental pathologies are not single but rather multiple in low birth weight deliveries. The prevalence of placental pathology in low birth weight infants is rather high and the examination of placenta provides considerable amount of data in such cases.

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