



## Original Research Article

## To correlate placental findings with maternal morbidities in all preterm and full term pregnancies

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## ABSTRACT

**Background & Methods :** This is a forthcoming kind of study and establishes the clinicopathological relationship of strange placental discoveries with pregnancy related maternal morbidities and related fetal result altogether preterm and full term pregnancies. Placentae from 80 conveyances of preterm and full term incubation were gathered from work room.

**Result:** The majority of cases (51.30%) in our study displayed low birth weight. Amongst the LBW neonates 35.50% of babies are associated with preterm deliveries and 14.47% were associated with full term pregnancy. Amongst the neonates with normal birth weight, 3.94% were associated with preterm labour and 46.05% were full term. There was no significant association between length of cord and period of gestation. The majority of cases showed eccentric attachment of cord with only 2.50% of preterm cases showing battledore insertion.

**Study Designed:** Observational Study.

**Conclusion:** The gross features of placenta varies significantly with period of gestation. Parameters such as weight, size, number of cotyledons and thickness of placenta decreases in preterm labour. The microscopic lesions observed in decreasing order of frequency were perivillous fibrin deposition (62.50%) followed by chorioamnionitis (26.25%), infarct (21.25%) and calcification (17.50%). Other pathological lesions includes villous oedema (12.50%) and retro placental hematoma (2.50%). All the histomorphological changes were conspicuous in preterm pregnancies. To prevent the maternal and child morbidity and mortality the community based health education, care for prevention of anaemia and personal hygiene must be promoted more vigorously in rural areas of our country.

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### 1. Introduction

The ovum is fertilized in the fallopian tube and reaches the uterine cavity as a 16-cell morula, which rapidly converts into a blastocyst. Its outer cell layer differentiates into the trophoblast, which completely envelopes the blastocyst and inner cell become inner cell mass which differentiates into embryo.<sup>1</sup>

Implantation occurs on the upper and posterior wall of the uterus.<sup>2</sup> The implanting blastocyst adheres to and penetrates the endometrium. By the 10th or 11th

post- ovulatory day, the ovum is totally embedded in the endometrial stroma, and the superficial endometrial epithelium has re- established its continuity.<sup>3</sup>

The placenta is an endocrine organ, as it plays a vital role in the transfer of oxygen and nutrients to the foetus, and it acts as an immunological barrier that prevents the rejection of the foetal allograft.<sup>4</sup> Exchange of gases such as oxygen, carbon dioxide, and carbon monoxide is accomplished by simple diffusion. Exchange of nutrients and electrolytes, such as amino acids, free fatty acids, carbohydrates and vitamins, is rapid and increases as pregnancy advances.

Immunological competence begins to develop late in the first trimester, by which time the foetus makes all of

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the components of complement.<sup>5</sup> Immunoglobulins consist almost entirely of maternal immunoglobulin G (IgG), which begins to be transported from mother to foetus at approximately 14 weeks. In this manner, the foetus gains passive immunity against various infectious diseases. Newborns begin to produce their own IgG, but adult levels are not attained until the age of 3 years.<sup>6</sup>

## 2. Materials and Methods

This is a prospective type of study and constitutes the clinicopathological correlation of abnormal placental findings with pregnancy related maternal morbidities and associated foetal outcome in all preterm and full term pregnancies. Placentae from 80 deliveries of preterm and fullterm gestation were collected from labour room.

For the study the cases were selected which fulfil the inclusion criteria such as all singleton pregnancies, all twin pregnancies, cases who presented with gestation period of <37 weeks, all patients with haemoglobin level of <11 gm/dl; cases associated with foetal abnormalities including low birth weight, IUGR and IUD.

We have excluded all the cases who had any associated systemic disorders like diabetes mellitus, preeclampsia, eclampsia, SLE, CNS disorders like epilepsy, poliomyelitis, polyneuritis and psychiatric disorders, rheumatic heart disease and pulmonary disorders like pneumonia and COPD; established cases of haemoglobinopathies and other haemoglobin disorders less anaemia and patients who were not willing to participate in study.

The cohort of patients with singleton uncomplicated term pregnancy, foetus with birth weight of ≥2.5 Kg, gestational period of >37 weeks, blood pressure < 140/90 mm of Hg throughout pregnancy and haemoglobin value of ≥11 g/dl formed the control group.

## 3. Results

**Table 1:** Gestational age group of neonates

Gestational Age	No of neonate	Percentage
<12 weeks	01	1.3
13-24 weeks	02	2.5
25-36 weeks	31	38.8
>36 weeks	46	57.5
Total	80	100

The bar diagram depicts the distribution of neonates as per gestational age. We found that the maximum number of neonates (57.50 %) were in the gestational age group of >36 weeks and 42.6% of neonates in gestational age group of 8-36 weeks.

The majority of cases (51.30%) in our study displayed low birth weight. Amongst the LBW neonates 35.50% of babies are associated with preterm deliveries and 14.47%

**Table 2:** Birth weight distribution

Birth weight (kg)	No of neonate	Percentage
<2.5 kg	33	41.3
>2.5 kg	41	51.3
Not Applied	06	7.5
Total	80	100

were associated with full term pregnancy. Amongst the neonates with normal birth weight, 3.94% were associated with preterm labour and 46.05% were full term.

**Table 3:** Placental pathological lesions

Placental Pathological Lesions	Frequency		Percentage
	Positive	Negative	
Chorioamnionitis	21	59	26.25%
Infarction	17	63	21.25%
Retro placental hematoma	2	78	2.50%
Perivillous fibrin	50	30	62.50%
Calcification	14	66	17.50%
Villous oedema	10	70	12.50%

There was no significant association between length of cord and period of gestation. The majority of cases showed eccentric attachment of cord with only 2.50% of preterm cases showing battledore insertion.

## 4. Discussion

A doubled risk of preterm delivery with anaemia during the second trimester but not during the third trimester. Likewise Allan LH reported the birth weights of 1.69, 2.75, and 3.56 for haemoglobin concentrations of 90–109, 70–89, and 110–119 g/L, respectively.<sup>7</sup> Thus Period of gestation is an important determinant of foetal birth weight. According to WHO, infants with birth weight of <2500 gms are considered as low birth weight babies.<sup>8</sup> The result of our study was in accordance with these authors.

In the morphological findings of placenta we have found decrease in weight and size of placenta with the increase in severity of anaemia.<sup>9</sup> The mean placental weight in patients of control group is 463 gms. Whereas in cases of mild, moderate and severe anaemia it is 439gms, 350 gms and 270 gms respectively. In a study by Mongia SM et al similar results were found. The mean placenta weight in mild, moderate and severe anaemia was 350gms, 300gms and 250gms.<sup>10</sup> Therefore our study was consistent with the observations.

On the contrary some authors have reported increase in the placental weight with increase in severity of maternal anaemia.<sup>11</sup> Beisehler and Godfrey hypothesized that maternal anaemia causes inadequate oxygenation of the fetoplacental unit and in term invokes physiological response resulting in compensatory placental hypertrophy

which is an adaptation to a physiological stress.<sup>12</sup> So conclusion is that due to mild to moderate anaemia in mother because of hypoxia compensatory placental hypertrophy occur. But in severe anaemia, placenta affected so much that it cannot go under compensatory hypertrophy.<sup>13</sup>

The mean placental size in the control group of this study was 16.7 cms and it decreases progressively with degree of anaemia. In a study done by Begum M, Ara S, Begum S et al. the mean placental size in control group was 15.60±0.7, whereas in cases of mild and moderate anaemia it was 18.04±1.3 and 18.80±1.9, which was contradictory to our study.<sup>14</sup>

## 5. Conclusion

The gross features of placenta varies significantly with period of gestation. Parameters such as weight, size, number of cotyledons and thickness of placenta decreases in preterm labour. The microscopic lesions observed in decreasing order of frequency were perivillous fibrin deposition (62.50%) followed by chorioamnionitis (26.25%), infarct (21.25%) and calcification (17.50%). Other pathological lesions includes villous oedema (12.50%) and retro placental hematoma (2.50%). All the histomorphological changes were conspicuous in preterm pregnancies. To prevent the maternal and child morbidity and mortality the community based health education, care for prevention of anaemia and personal hygiene must be promoted more vigorously in rural areas of our country.

## 6. Conflicts of Interest

All contributing authors declare no conflicts of interest.

## 7. Source of Funding

None.

## References

1. Benirschke K, Kaufmann P. Pathology of the Human Placenta. New York: Springer Verlag; 1995.
2. Fox H. Pathology of the placenta, Voll 7 in the series Major Problem in Pathology. Philadelphia W B Sand: Co. Ltd; 1978.
3. Gresell DJ, Kraus FT. Diseases of the placenta, Blaustein's Pathology of the female Genital Tract. Springer-Verlag; 1994.
4. Kurman RJ, Main CS, Chen HC. Intermediate trophoblast: a distinctive form of trophoblast with specific morphological, biochemical and functional features. *Placenta*. 1984;5(4):349-70. doi:10.1016/s0143-4004(84)80015-6.
5. Konar H, D C Dutta The Placenta and Foetal Membranes. Textbook of obstetrics. In: 8th Revised Edn.. vol. 6. Kolkata; 2004. p. 28-39.
6. Pijnenborg DR. Trophoblastic invasion of the Human Decidua from 8 to 18 weeks of pregnancy. *Placenta*. 1980;1:3-19.
7. Kurman RJ. The morphology, biology and pathology of intermediate trophoblast: A look back to the present. *Hum Pathol*. 1991;22:847-58.
8. Pijnenborg D. Uteroplacental arterial changes related to interstitial trophoblast migration in early human pregnancy. *Placenta*. 1983;4:397-414.
9. Honjoh Y, Nishimura Y, Kanomata. Morphological studies on the placenta. *Kobe J Med Sci*. 1994;40(1):1-11.
10. Fox H. Perivillous fibrin deposition in the human placenta. *Am J Obstet Gynecol*. 1967;98(2):245-51. doi:10.1016/s0002-9378(16)34594-x.
11. Schuler-Maloney D, Lee S. The Placenta- To know me is to love me. A reference guide for gross placental examination. DSM Path Works, Inc; 1998.
12. Sebire NJ, Jauniaux E. Fetal and placental malignancies: prenatal diagnosis and management. *Ultrasound Obstet Gynecol* . 2009;33(2):235-44. doi:10.1002/uog.6246.
13. Kumar V, Abbas A, Fausto N, Aster J. Robbins and Cotran Pathologic Basis of Disease. In: and others, editor. 8th Edn.. vol. 8 of 22. Elsevier; 2010. p. 1005-64.
14. Lurain J. Gestational trophoblastic disease I: epidemiology, pathology, clinical presentation and diagnosis of gestational trophoblastic disease, and management of hydatidiform mole. *Am J Obstet Gynecol*. 2010;203(6):531-9.

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