

Original Article

Placental Pathology in Severe Pre-eclampsia and Eclampsia

Ojha Kamala¹, Rawal Suniti¹, Jha Abhimanyu²

¹Department of Obstetrics and Gynaecology, Institute of Medicine, Kathmandu, Nepal.

²Department of Pathology, Institute of Medicine, Kathmandu, Nepal.

ABSTRACT

Introduction: Hypertensive disorders complicating pregnancy contribute significantly to maternal and perinatal morbidity and mortality. Since placenta is the functional unit between the mother and fetus examination of placenta can give an idea about prenatal experience of fetus. The aim is to observe the morphology and histopathology of placenta in pregnancy with severe preeclampsia / eclampsia between 20-42 weeks of gestation.

Materials and Methods: This was a prospective, descriptive study carried out in the Department of Obstetrics and Gynaecology and Department of Pathology at Institute of Medicine, Tribhuvan University Teaching Hospital, TUTH for one year, starting from 15th May 2015 - 14th May 2016. A total 55 placentas, 48 of severe preeclampsia and 7 of eclampsia were collected and placental morphometric parameters, gross and histopathological features were examined.

Results: It was found that placental morphometric parameters were significantly reduced. Histopathological study showed significant number of syncytial knots, areas of fibrinoid necrosis, hyalinization and calcification. These placental findings were associated with significantly decreased weight of fetus at birth.

Conclusions: Preeclampsia and eclampsia cause significant placental morphometric and histological changes which in turn adversely affects neonatal birth weight.

Key words: Eclampsia; Fibrinoid; Necrosis; Syncytial knots; Placenta, Preeclampsia

Copyright: This is an open-access article distributed under the terms of the Creative Commons Attribution 4.0 International License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Correspondence:

Dr. Abhimanyu Jha, MD
Professor, Department of Pathology
Tribhuvan University Teaching Hospital,
Maharajgunj, Kathmandu, Nepal
ORCID ID: 0000-0003-4477-6129
Email: jhaabhimanyu@yahoo.com

Submitted: 2nd February 2018

Accepted: 16th April 2018

Published: 1st June 2018

Sources of Support: None

Conflict of Interest: None



Citation: Ojha K, Rawal S, Jha A. Placental Pathology in Severe Pre-eclampsia and Eclampsia. Nep Med J. 2018;1:32-5. DOI: 10.3126/nmj.v1i1.20397

INTRODUCTION

Hypertensive disorders complicate 5-10% of all pregnancies, and contribute greatly to maternal morbidity and mortality. Of these disorders, the preeclampsia syndrome, either alone or superimposed on chronic hypertension (HTN) is the most dangerous.¹

Placenta has been described as a “diary of intrauterine life”.² Pregnancy complications like HTN are reflected in the placenta in a significant way (both macroscopically and microscopically). The inadequate trophoblast invasion of maternal spiral arteries leading to decreased placental perfusion is associated with the changes seen in the placenta of pre-eclamptic women.³ Foetal growth is essentially dependent on formation and full development of the placenta. As placenta is a mirror which reflects the intrauterine

status of the foetus, examination of placenta gives a clear idea of what had happened with it, when it was in the mother’s womb and what is going to happen with the foetus in future.^{4,5}

How pregnancy incites or aggravates HTN remains unsolved despite decades of intensive research. As there is relationship between abnormal placentation and preeclampsia (PE) a thorough study of placenta is indispensable to evaluate possible etiological factor. Despite observed link between placenta and newborn health, examination of placenta is seldom performed in institutions, so the present study was carried out to analyze and study the morphometric features, gross and histological changes of placenta in severe preeclampsia and eclampsia.

MATERIALS AND METHODS

The study was a prospective, descriptive study, conducted in the Department of Obstetrics and Gynaecology and Department of Pathology, Tribhuvan University Teaching Hospital, TUTH in Kathmandu, Nepal for one year from 15th May 2015 - 14th May 2016. Permission was obtained from institutional review board to conduct the study. All singleton pregnancies (live or IUD) between 20-42 weeks with severe preeclampsia/eclampsia were included in the study, exclusion criteria being multiple pregnancies, pregnancies with GDM, vascular disorders, renal disease, connective tissue disease, chronic HTN, heart disease and congenital malformations.

The placentas from both vaginal and cesarean deliveries were included. Immediately after the delivery, weight (wt) of the baby was taken. Once the placenta was delivered, it was washed in the running tap water. Any abnormality of the placenta and cord was noted. Umbilical cord was cut at its placental site of insertion and placental wt was measured in grams. As the shape of placenta is not always circular, diameter was taken along two axes perpendicular to each other and average diameter was taken. Placental area was calculated using the following formula: $Area = \pi r^2$. Placental volume was measured by using the water displacement technique. Gross examination of placenta was done for the presence of infarction, calcification, retroplacental hematoma and chorangioma. These findings were confirmed by the pathologist once the placenta was delivered to pathology department. Placenta was preserved in 10% formalin solution and then transported to Department of Pathology. Samples were taken from the insertion of umbilical cord, margins 3, 6, 9, 12 O'clock positions, centre of the placenta, fibrotic, calcified and infarcted area and histopathological examination was done.

RESULTS

There were 4276 deliveries during the study period of which 62 were severe preeclampsia (prevalence 1.45%) and 7 were eclampsia (prevalence 0.16%). Out of 62 PE cases, only 48 meeting the inclusion criteria and all 7 cases of eclampsia were included in the study.

The placental changes were studied under the headings-placental morphometry (Table 1), gross anatomy (Table 2) and histopathology which included villous pathology (Table 3) and stromal pathology (Table 4). Average weight of placenta was 386 gm ranging from 150 gm to 500 gm. Similarly there was a wide range of placental area ranging from 92.4 cm² to 240.4 cm²

Grossly various findings were found in the placenta. Among them, infarction was seen in 12 cases (21.82%) followed by calcified area comprising of 11 (20.00%) cases (table 2). Microscopically syncytial knots were the most common findings (n=53; 96.36%) followed by fibrinoid necrosis (n=18; 32.72% table 3).

In the stroma of the placenta, calcification was the most common pathology (n=52; 94.54%) followed by hyalinised areas (n=9; 16.36%). Endothelial proliferation was seen in 6(10.91%) cases. (Table 4) There was wide variation in birth weight ranging from 0.8-3.5 kg with mean birth weight of 2.37 kg. The mean fetoplacental ratio was 6.1. (Table 5)

Table 1: Placental Morphometric Study

Parameters	Mean	SD	Minimum	Maximum
Placental Wt (gm)	386.36	87.11	150	500
Placental Area (cm ²)	148.57	35.87	92.41	240.41
Placental Volume (cc)	353.18	69.56	225	475

Table 2: Gross Anatomy of Placenta

Gross findings	Number of cases
Infarcted area	12 (21.82%)
Calcified area	11 (20%)
Marginal insertion of cord	3 (5.45%)
Retroplacental hemorrhage	3 (5.45%)
Chorangioma	1 (1.82%)
Normal	34 (61.82%)

Similarly effect of anti hypertensive drugs on placental change was studied. Out of 55 patients included in the study, 45 were on anti hypertensive drugs and all 45 placentas showed histopathological changes significant to PE despite the duration and type of drug. There was also no difference in the placental changes of patients taking 500 mg or 1000 mg of calcium/day. Similarly 4 patients were on Aspirin 75 mg/day in addition to anti hypertensive drugs, who still showed placental changes.

DISCUSSION

Placenta is a vital organ maintaining pregnancy and promoting fetal development. A fetus derives its nutritional substances and obtains its metabolic and immunological requirements from the placenta. The impaired placental function in terms of abnormal placental morphology or histology accounts for the fetal and neonatal complications seen in pregnancies complicated by severe preeclampsia and eclampsia.

Incidence of severe PE was 1.45% and that of eclampsia was 0.16% which is higher than that reported by Gautam SK et al in a study conducted at the same institute in 2012.⁶ Higher incidence of eclampsia was noted in studies conducted at Paropakar Maternity and Women's Hospital and Patan Hospital (0.29% and 0.24% respectively).^{7,8}

Placental Morphometry

Placental weight: In our study the mean wt of the placenta was 386.36 gm similar to that observed by Kartha et al⁵. All the placentae from severe pre-eclampsia weighed < 500gm, the least wt recorded being 150 gm similar to a study done by Narasimha and Vasudeva.⁹ From all these results it can be inferred that the placental weight is reduced in pregnancies complicated by hypertension which could be due to reduced uteroplacental blood flow.

Area of Placenta: Mean placental area was 148.57 cm² similar to that noted by Udaina A et al.¹⁰ Unlike our study Vaibhav et al¹¹ and Ghodke S.P. et al¹² noted higher values, 182.84 +/- 56.71 cm² and 185.04 +/- 33.72 cm² respectively. The lower placental area in our study compared to these studies could be due to the big difference in gestational age at delivery, ranging from 28 weeks to 40 weeks 2 days.

Table 3: Villous Pathology

Features	Number of cases
Syncytial knots	53 (96.36%)
Fibrinoid necrosis	18 (32.72%)
Hyalinised villi	7 (12.72%)
Acute atherosclerosis	0

Table 4: Stromal Pathology

Features	Number of cases
Syncytial knots	53 (96.36%)
Fibrinoid necrosis	18 (32.72%)
Hyalinised villi	7 (12.72%)
Acute atherosclerosis	0

Table 5: Fetal weight and Feto-Placental Ratio

	Mean	SD	Minimum	Maximum
Birth Wt in kg	2.37	0.65	0.8	3.5
Feto-placental ratio	6.1	0.96	3.2	8.7
Placental Volume (cc)	353.18	69.56	225	475

Volume of Placenta: We found average placental volume of 353cc.¹⁸ Slightly higher value was noted in studies done by Majumdar et al³ Vaibhav et al¹¹ and Kartha et al⁵ This may be because these three studies included all PIH cases in contrast to our study which included only severe PE and eclampsia cases.

Gross Anatomy of Placenta

Site of Umbilical cord insertion: Abnormal cord insertion seems to be associated with impaired development and function of the placenta, and has been linked to PIH.¹³ We found marginal cord insertion in 5.45 % of cases similar to a study done by Vijayalaxmi et al.¹⁴ This finding was much less than that found by Majumdar et al in which incidence was 20%.³

Infarction: Extensive placental infarction is usually seen in placentae from preeclamptic mothers when there is a vascular abnormality which predisposes to thrombosis.¹⁵ Infarction was the most common gross anatomical abnormality, found in our study (21.82%) similar to that by Kartha et al.⁵ Incidence was 48% and 53.1 % in studies by Vijayalaxmi et al¹⁴ and Salgado et al.¹⁶

Calcification: Calcification is regarded as evidence of placental senescence or degeneration. We found calcification in 20% cases. Slightly higher incidence was noted by Vassiliki et al (32.32%)¹⁷ and Vijayalaxmi et al (35%).¹⁴ This higher incidence could be because these two studies included only late preterm and term cases in contrast to our study which included early preterm cases as well.

Retro-placental Hematoma: Retroplacental hematoma was observed in 5.45% of cases which was low compared to the study by Narasimha and Vasudeva.⁹ Incidence was quite high i.e. 38.88% in severe PE cases in a study by Tangirala et al.¹⁸

Chorangioma: Chorangioma is the hemangioma of placenta and was observed in only 1 case of severe PE (1.82%) which was less than that noted by Kartha et al.⁵ In a study by Ogino and Redline incidence was 0.51% and it was more commonly associated with PE, multiple gestation and prematurity.¹⁹

Villous Pathology

Syncytial Knots: Increased syncytial knots are associated with conditions of uteroplacental malperfusion. We found syncytial knots in 96.36% of cases, which is quite higher than that noted by Majumdar et al³ and Kartha et al⁵ Masodkar et al²⁰ and Avasthi et al²¹ found this finding in 69% and 80% cases of PIH respectively. Syncytial knot formation being an indication of severity of PE could have been seen more in our study due to inclusion of cases of severe PE and eclampsia only.

Fibrinoid Necrosis: Fibrinoid necrosis of placental villi is a highly characteristic lesion, which is due to replacement of the villus by fibrin.²² In the present study incidence was 32.72% which is higher than that noted by Narasimha and Vasudeva⁹ and Kartha et al.⁵

Acute Atherosclerosis: Acute atherosclerosis is associated with severe and early PE. The frequency of acute atherosclerosis was 10.2% in PE in the study by Kim YM et al²³ Kartha et al noted slightly high incidence (16%) of acute atherosclerosis.⁵ However none of the cases showed acute atherosclerosis in the index study probably due to very small sample size as compared to these studies.

Hyalinised Villi: Hyalinization is villous hypovascularity, mechanism of which is not fully understood. Hyalinised villi was observed in 12.72% cases. Similar finding was reported by Majumdar et al³ In contrast Kartha et al noted hyalinised villi in significantly high number of cases (27%).⁵

Stromal Pathology

Fibrosis: Fibrosis was not seen in any of the cases in the present study. However various other studies have shown presence of fibrosis in PIH. Kartha et al found 38% cases of stromal fibrosis⁵ whereas it was observed in 92% cases of toxemia in the study by Narasimha and Vasudeva.⁹

Calcification: Incidence of stromal calcification was high in the present study (94.54%) whereas Kartha et al and Vijayalaxmi et al reported quite low incidence of 25% and 35% respectively.^{5,24}

Hyalinised Areas: Hyalinised areas was observed in 7 cases (12.72%). This finding is consistent with the study by Majumdar et al³ In contrast Kartha et al noted hyalinised areas in 28% of cases which was more than double the index study, which could be attributed to the larger sample size of the study.⁵

Endothelial Proliferation: In the present study endothelial proliferation was found in 10.91% of cases which is similar to that noted by Kartha et al.⁵

Fetal Weight and Feto-Placental Ratio

Fetal Weight: Mean birth wt was noted to be 2.37 kg. This finding corroborates with the studies by Madhu L et al²⁵ and Udaina and Jain²⁶ with average neonatal wt of 2.1 and 2.2 kg respectively. With reference to these values it is inferred that the wt of a newborn baby is significantly low in pregnancy induced HTN due to placental insufficiency.

Feto-Placental Ratio: Also known as placental co-efficient, feto-placental weight ratio is often used an index of placental nutritional efficacy and is related to adverse perinatal outcomes. We found mean feto-placental ratio of 6.1 which was in concordance with the study by Nag U et al²⁷ and Gugapriya et al²⁸ Increased mean feto-placental ratio in hypertensive pregnancies compared to normotensive cases was also observed by Majumdar et al³ and

Kartha et al⁵ So it can be concluded that the foeto-placental wt ratio is increased in PIH and that the placental wt is more severely affected than that of the foetal wt.

Antihypertensive Drugs

Although 45 patients out of 55 were on anti hypertensive drugs, all 45 placentas showed histopathological changes significant to preeclampsia.

Aspirin/Calcium: Four patients who had history of preeclampsia in previous pregnancy were kept on Aspirin 75 mg/day, who still showed placental changes. Similarly all patients took calcium, 40 took 500 mg/day and 15 took 1000 mg/day, however the histological changes were more or less similar in both groups.

REFERENCES

- Cunningham FG, Leveno KJ, Bloom SL, Spong CY, Dashe JS, Hoffman BL, et al. editors. Williams Obstetrics. 24th ed. New York: McGraw Hill; 2014. pp.728-70. [Crossref](#)
- Redline RW. Placental pathology: A systematic approach with clinical correlations. *Placenta*. 2008; 29(Suppl A):S86–S91. [Crossref](#)
- Majumdar S, Dasgupta H, Bhattacharya K, Bhattacharya A. A study of placenta in normal and hypertensive pregnancies. *J. Anat. Soc. Ind.* 2005;54:1-9.
- Londhe PS, Mane AB. Placental Morphometry in Relation to Birth Weight of Full Term Newborn Babies. *NJIRM*. 2012;3:67-72.
- Kartha S, Poothiade U, Jayalakshmy PS. Placental Pathology in Pregnancy Induced Hypertension. *Journal of Evolution of Medical and Dental Sciences* 2014;3:9272-8. [Crossref](#)
- Gautam SK, Paudel K, Silwal K. Management and Outcome of Pre-eclampsia/Eclampsia among patient admitted in maternity ward in tertiary hospital. *JIOM*. 2013;35:230-7
- Choudhary P. Eclampsia: a hospital based retrospective study. *Kathmandu Univ Med J*. 2003;1:237-41.
- Acharya G, Schultz S. Eclampsia in Patan hospital: A two year retrospective study. *J Nepal Med Assoc*. 1991;29:254-8.
- Narasimha A, Vasudeva DS. Spectrum of changes in placenta in toxemia of pregnancy. *Indian J Pathol Microbiol* 2011;54:15-20. [Crossref](#)
- Udainia A, Bhagwat SS, Mehta CD. Relation between placental surface area, infarction and fetal distress in pregnancy induced hypertension with its clinical relevance. *J Anat Soc Ind.* 2004;53:27-30.
- Anjankar VP, Parchand MP, Anjankar AP, Kamdi NY, Meshram MM. Placental Morphometry in Toxaemia of Pregnancy. *Sch J App Med Sci* 2014;2:205-8.
- Ghodke SP, Kale NV. Hypertension Affecting Morphometry of Placenta. *Med Pulse – Int Med J*. 2014;1:27-9.
- Ebbing C, Kiserud T, Johnsen SL, Albrechtsen S, Rasmussen S. Prevalence, Risk Factors and Outcomes of Velamentous and Marginal Cord Insertions: A Population-Based Study of 634,741 Pregnancies. *Sahasrabuddhe V, ed. PLoS One*. 2013; 8(7):e70380.
- Vijayalakshmi B, Kittali S. Morphological changes of placenta in cases of pre-eclampsia and perinatal outcome. *Int J Sci Stud*. 2015;3:137-42.
- Khong TY. Acute atherosclerosis in pregnancies complicated by hypertension, small for gestational age infants and diabetes mellitus. *Arch Pathol Lab Med*. 1991;115:722-5. [Crossref](#)
- Salgado SS, Pathmeswaran A. Effects of placental infarctions on the fetal outcome in pregnancies complicated by hypertension. *J Coll Physicians Surg Pak*. 2008;4:213-6.
- Krielessi V, Papantoniou N, Papageorgiou I, Chatzipapas I, Manios E, Zakopoulos N, Antsaklis A. Placental pathology and blood pressure's level in women with hypertensive disorders in pregnancy. *Obstet Gynecol Int*. 2012;684083. [Crossref](#)
- Tangirala S, Kumari D. Placental morphology in hypertensive disorders and its correlation to neonatal outcome. *IAIM*. 2015;2:35-8.
- Ogino S, Redline RW. Villous capillary lesions of the placenta: distinctions between chorangioma, chorangiomatosis, and chorangiosis. *Hum Pathol*. 2000;31:945-54. [Crossref](#)
- Masodkar AR, Kalamkar LR, Patki PS. Histopathology of placenta and its correlation with foetal outcome. *J Obstet Gynaecol India*. 1985;35:294-300.
- Avasthi K, Micha U, Sabharwal BD. Histopathology of placenta and its correlation with foetal outcome. *J Obstet Gynaecol India*. 1991;41:317-28.
- Dhabha P, Gupta G, Bapna N. Histological Study of Human Placenta In Normal And Pregnancy Induced Hypertension (P I H) Cases. *Int J Pharm Sc Invent*. 2013;2:30-5.
- Kim YM, Chaemsaithong P, Romero R, Shaman M, Kim CJ, Kim JS et al. The Frequency of Acute Atherosclerosis in Normal Pregnancy and Preterm Labor, Preeclampsia, Small for Gestational Age, Fetal Death and Midtrimester Spontaneous Abortion. *J Matern Fetal Neonatal Med*. 2015;28:2001-09. [Crossref](#)
- Vijayalakshmi B, Sunitha Kitteli. A Study of Histopathological Changes of Placenta in Pre Eclampsia and Perinatal Outcome. *Journal of Evolution of Medical and Dental Sciences*. 2015;4:11667-73. [Crossref](#)
- Madhu L, Karthavya SL, Lepakshi BG. Histomorphological and morphometrical changes of placental terminal villi of normotensive and hypertensive mothers. *Int. J Med Sc Pharm Res*. 2015;1:1-14.
- Udaina A, Jain ML. Morphological Study of Placenta in Pregnancy Induced Hypertension with its Clinical Relevance. *J Anat Soc Ind.* 2001;50:24-7.
- Nag U, Chakravarthy VK, Rao DR. Morphological changes in placenta of hypertensive pregnant women. *Ind J Res Rep Med Sci*. 2013;3:1-4.
- Gugapriya TS, Singh S. Microanatomical analysis of hypertensive placenta: a retrospective case control study. *National Journal of Clinical Anatomy*. 2013; 2(1):5-10.

CONCLUSIONS

Severe PE and eclampsia has definite adverse influence on placental architecture. The morphological and histological changes in placenta compromise utero-placental blood flow and significantly reduce the neonatal birth wt. As placenta is the bridge between mother and fetus, placental examination can provide a valuable insight into the mechanism of placental dysfunction and identify conditions with recurrence, resulting in counseling and management of subsequent pregnancies.