ISSN: 2581 – 3633

International Journal of Medical Science and Advanced Clinical Research (IJMACR)

Available Online at: https://www.ijmacr.com

Volume – 9, Issue – 5, September – 2026, Page No.: 385 – 394

A Study of Histopathological Changes of Placenta in Pre-Eclampsia and Eclampsia

[1]Dr. Pacharne Bhakti Jaysingh, MBBS, MS, OBGY, Department of Obstetrics and Gynaecology, Bharati Vidyapeeth Deemed to be University Medical College & Hospital, Sangli, Maharashtra, India.

[2]Dr. Manjusha Wagh, Assistant Professor, Department of Obstetrics and Gynaecology, Bharati Vidyapeeth Deemed to be University Medical College & Hospital, Sangli, Maharashtra, India.

Corresponding Author: Dr. Pacharne Bhakti Jaysingh, MBBS, MS, OBGY, Department of Obstetrics and Gynaecology, Bharati Vidyapeeth Deemed to be University Medical College & Hospital, Sangli, Maharashtra, India.

How to citation this article: Dr. Pacharne Bhakti Jaysingh, Dr. Manjusha Wagh, "A Study of Histopathological Changes of Placenta in Pre-Eclampsia and Eclampsia", IJMACR– September– 2026, Volume – 9, Issue – 5, P. No. 385 – 394.

Type of Publication: Original Research Article

Conflicts of Interest: Nil

Abstract

Background: Hypertensive disorders of pregnancy, particularly pre-eclampsia and eclampsia, are major causes of maternal and perinatal morbidity and mortality. The placenta is a key organ affected by these disorders, and Histopathological examination may reveal characteristic changes associated with impaired uteroplacental perfusion and maternal vascular dysfunction.

Aim: To study the Histopathological changes of the placenta in pre-eclampsia and eclampsia and evaluate their association with neonatal outcomes.

Materials and Methods: A prospective observational study was conducted in the Department of Obstetrics and Gynaecology at a tertiary care centre over 18 months (January 2024 to May 2025). A total of 66 patients were included, comprising 33 cases of pre-eclampsia and 33 cases of eclampsia. Placental specimens were collected after delivery, fixed in 10% neutral buffered formalin, processed, and examined microscopically after hematoxylin and eosin staining. Histopathological findings, including micro calcification, infarction, stromal fibrosis, decidual vasculopathy, immature villi, chorio amnionitis, and chorangios is, were evaluated. Data were analysed using SPSS version 24.0, with $p<0.05$ considered statistically significant.

Results: The mean maternal age was comparable between the eclampsia and pre-eclampsia groups (25.3 ± 5.3 years and 26.1 ± 5.1 years, respectively). The mean placental weight and fetoplacental ratio showed no significant difference between groups. Micro calcification was the most common Histopathological lesion, observed in 80% of cases, followed by infarction (73%) and immature villi (68%). Micro calcification (88% vs 73%), infarction (76% vs 70%), decidual vasculopathy (61% vs 45%), stromal fibrosis (27% vs

15%), and immature villi (73% vs 64%) were more frequent in eclampsia compared with pre-eclampsia; however, none of these associations reached statistical significance. No significant association was observed between placental lesions and preterm birth or low birth weight.

Conclusion: Placental Histopathological abnormalities are common in pregnancies complicated by pre-eclampsia and eclampsia, reflecting underlying maternal vascular and ischemic changes. Although lesions were more frequent in eclampsia, statistical significance was not demonstrated. Placental examination remains a valuable tool for understanding the pathological basis of hypertensive disorders of pregnancy and their impact on fetal outcomes.

Keywords: Pre - Eclampsia, Eclampsia, Placenta, Histopathology, Decidual Vasculopathy, Infarction, Micro Calcification.

Introduction

Pregnancy-induced hypertensive disorders remain among the leading causes of maternal and perinatal morbidity and mortality worldwide. These disorders include gestational hypertension, pre-eclampsia, and eclampsia, with pre - eclampsia and eclampsia representing severe forms associated with significant maternal and fetal complications. Pre-eclampsia is characterized by the development of hypertension after 20 weeks of gestation along with proteinuria or evidence of maternal organ dysfunction, whereas eclampsia is defined by the occurrence of generalized tonic-clonic seizures in a woman with pre-eclampsia without another neurological cause. Despite extensive research, the exact pathogenesis of these disorders remains incompletely understood; however, abnormal placentation and defective uteroplacental vascular remodeling are considered central mechanisms.[1,2]

The placenta plays a crucial role in maintaining pregnancy by facilitating maternal–fetal exchange and regulating fetal growth. Abnormal placental development and impaired trophoblastic invasion result in inadequate spiral artery transformation, reduced uteroplacental perfusion, and placental ischemia. These pathological changes contribute to the release of anti angiogenic factors, inflammatory mediators, and endothelial dysfunction, which are responsible for the clinical manifestations of pre-eclampsia and eclampsia.[3]

Histopathological examination of the placenta provides valuable insight into the underlying disease mechanisms and reflects the effects of maternal vascular disorders on placental structure. Placental lesions commonly associated with hypertensive disorders of pregnancy include decidual vasculopathy, infarction, increased syncytial knots, accelerated villous maturation, stromal fibrosis, micro calcification, chorangios is, and inflammatory lesions.[4,5] These changes may adversely affect fetal growth and contribute to complications such as fetal growth restriction, prematurity, low birth weight, and perinatal mortality.

Decidual vasculopathy is considered a characteristic placental lesion associated with pre-eclampsia and represents the morphological manifestation of abnormal maternal vascular response. It includes acute atherosis, fibrinoid necrosis, and mural hypertrophy of decidual vessels. Placental infarction results from impaired maternal blood flow and is frequently observed in severe hypertensive disorders. Similarly, accelerated villous maturation reflects placental adaptation to chronic hypoxia caused by reduced maternal perfusion.[6]

Eclampsia represents the extreme end of the spectrum of hypertensive disorders of pregnancy and is associated with more severe maternal endothelial dysfunction and placental abnormalities compared with pre-eclampsia. However, the relationship between the severity of hypertensive disease and specific placental histological lesions remains variable across studies. Some investigators have demonstrated a higher frequency of infarction, decidual vasculopathy, and villous abnormalities in eclampsia, whereas others have reported overlapping findings between pre-eclampsia and eclampsia.[7],[8]

The evaluation of placental pathology may therefore serve as an important tool for understanding disease progression, predicting neonatal outcomes, and identifying pregnancies affected by abnormal placentation. Although several studies have investigated placental changes in hypertensive disorders, variations exist regarding the prevalence and clinical significance of individual lesions. Therefore, the present study was undertaken to evaluate the Histopathological changes of placenta in patients with pre-eclampsia and eclampsia and to assess their association with neonatal outcomes.

Materials and Methods

Study Design and Setting

A prospective observational study was conducted in the Department of Obstetrics and Gynaecology at a tertiary care centre over a period of 18 months, from January 2024 to May 2025.

Study Population

The study included pregnant women diagnosed with pre-eclampsia or eclampsia who fulfilled the eligibility criteria and were admitted to the Department of Obstetrics and Gynaecology during the study period.

Inclusion Criteria

- Patients diagnosed with pre-eclampsia.
- Patients diagnosed with eclampsia.

Exclusion Criteria

Patients were excluded if they had:

- A history of chronic hypertension.
- A history of liver disease, hemolytic disease, diabetes mellitus, or renal dysfunction.
- Twin pregnancy.

Sample Size and Sampling Technique

The minimum sample size was calculated using the following formula:

$N = (Z\alpha/2)^2 \times P \times Q / E^2$

where:

- N = Sample size
- $Z\alpha/2 = 1.96$ at 95% confidence level
- P = 0.92 (based on the study by Panday et al., which reported placental micro calcification in 92% of patients with pregnancy-induced hypertension)
- $Q = 1 - P = 0.08$
- E = Allowable error (7%)

Accordingly, the calculated minimum sample size was 57.7, which was rounded to 58. During the study period, 66 consecutive patients were enrolled, comprising 33 cases of pre-eclampsia and 33 cases of eclampsia.

Data Collection

Data were collected using a predesigned semi-structured study proforma. Information recorded included demographic characteristics, socioeconomic status, menstrual and obstetric history, clinical examination findings, laboratory investigations, placental Histopathological findings, and neonatal outcomes.

Diagnostic Criteria

The diagnosis of hypertensive disorders of pregnancy was made according to the following criteria:

- **Pregnancy-induced hypertension (PIH):** Blood pressure ≥140/90 mmHg occurring after 20 weeks of gestation without proteinuria.
- **Pre-eclampsia:** Pregnancy-induced hypertension associated with new-onset proteinuria of ≥ 300 mg in a 24 - hour urine collection.
- **Eclampsia:** Pre - eclampsia complicated by generalized tonic-clonic convulsions not attributable to any other cause.

Placental Histopathological Examination

Following delivery, placental tissue was collected from each patient. The specimens were immediately fixed in 10% neutral buffered formalin, ensuring that the volume of fixative was at least ten times the volume of the tissue to achieve adequate fixation.

After fixation, each placenta was examined grossly for weight, thickness, diameter, calcifications, infarctions, and abnormalities of the umbilical cord and its insertion. Representative tissue sections were obtained from the maternal and fetal surfaces of the placenta, as well as the umbilical cord.

Paraffin-embedded tissue blocks were prepared, and 5-µm-thick sections were cut using a microtome. The sections were stained with routine hematoxylin and eosin (H&E) stain and examined under light microscopy. Histopathological evaluation included assessment for:

- Micro calcifications
- Stromal fibrosis
- Infarction
- Decidual vasculopathy
- Accelerated villous maturation
- Acute chorioamnionitis
- Chorangiosis
- Immature villi

Neonatal Outcome Assessment

Neonatal outcomes recorded included:

- Birth weight
- Apgar score at 1 minute
- Apgar score at 5 minutes
- Perinatal mortality
- Presence of major congenital anomalies

Statistical Analysis

Data were entered and analysed using Statistical Package for the Social Sciences (SPSS) version 24.0. Quantitative variables were expressed as mean ± standard deviation (SD), whereas categorical variables were presented as frequency and percentage.

Associations between categorical variables were assessed using the Chi-square test, while continuous variables were compared using the Student's t-test. A P-value <0.05 was considered statistically significant.

Ethical Considerations

The study protocol was approved by the Institutional Ethics Committee before commencement and was conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants before enrolment. Participation in the study involved no additional financial burden or harm to the patients, and confidentiality of patient information was maintained throughout the study.

Results and Observations

A total of 66 patients were included in the study, comprising 33 patients with eclampsia and 33 patients with pre-eclampsia.

Table 1: Baseline characteristics of the study population according to diagnosis

Variable	Eclampsia (n=33)	Pre-eclampsia (n=33)	Total (n=66)
Number of patients	33 (50%)	33 (50%)	66 (100%)
Age 19–25 years	20 (61%)	20 (61%)	40 (61%)
Age 26–35 years	12 (36%)	10 (30%)	22 (33%)
Age >35 years	1 (3%)	3 (9%)	4 (6%)
Mean age (years)	25.3 ± 5.3	26.1 ± 5.1	—

Observation: Both groups had an equal number of patients. The majority (61%) belonged to the 19–25 years age group. Mean age was comparable between the two groups.

Table 2: Comparison of placental weight and fetoplacental ratio between study groups

Variable	Eclampsia (n=33)	Pre-eclampsia (n=33)
Mean placental weight (g)	397.0 ± 62.3	399.7 ± 125.5
Range (g)	290–510	80–660
Mean fetoplacental ratio	4.8 ± 1.3	4.7 ± 1.6
Range	2.4–8.6	2.5–8.2

Observation: Mean placental weight and fetoplacental ratio were comparable in both groups, although placental weight showed greater variability in pre-eclampsia.

Table 3: Overall distribution of placental Histopathological findings (n=66)

Histopathological finding	Frequency	Percentage
Micro calcification	53	80%
Infarction	48	73%
Immature villi	45	68%
Decidual vasculopathy	35	53%
Stromal fibrosis	14	21%
Chorioamnionitis	14	21%

Observation: Micro calcification was the commonest Histopathological finding, followed by infarction and immature villi.

Table 4: Association of placental microcalcification and infarction with diagnosis

Histopathological finding	Eclampsia n (%)	Pre-eclampsia n (%)	p-value
Micro calcification	29 (88%)	24 (73%)	0.12
Infarction	25 (76%)	23 (70%)	0.58

Observation: Micro calcification and infarction were more frequent in eclampsia than pre-eclampsia; however, neither association was statistically significant.

Table 5: Association of stromal fibrosis and chorioamnionitis with diagnosis

Histopathological finding	Eclampsia n (%)	Pre-eclampsia n (%)	p-value
Stromal fibrosis	9 (27%)	5 (15%)	0.22
Chorioamnionitis	6 (18%)	8 (24%)	0.54

Observation: Stromal fibrosis was relatively more common in eclampsia, whereas chorioamnionitis was slightly more frequent in pre-eclampsia. Neither difference was statistically significant.

Table 6: Association of decidual vasculopathy and immature villi with diagnosis

Histopathological finding	Eclampsia n (%)	Pre-eclampsia n (%)	p-value
Decidual vasculopathy	20 (61%)	15 (45%)	0.21
Immature villi	24 (73%)	21 (64%)	0.42

Observation: Decidual vasculopathy and immature villi were more common among eclampsia patients, but the associations were not statistically significant.

Table 7: Association of placental histopathological findings with preterm birth

Histopathological finding	No Preterm (n=14)	Preterm (n=52)	p-value
Micro calcification	12 (86%)	41 (79%)	0.56
Infarction	10 (71%)	38 (73%)	0.91
Stromal fibrosis	4 (29%)	10 (19%)	0.44
Chorioamnionitis	3 (21%)	11 (21%)	0.98
Decidual vasculopathy	9 (64%)	26 (50%)	0.34
Immature villi	8 (57%)	37 (71%)	0.31

Observation: Although micro calcification, infarction and immature villi were frequently observed in preterm deliveries, none of the placental Histopathological findings showed a statistically significant association with preterm birth.

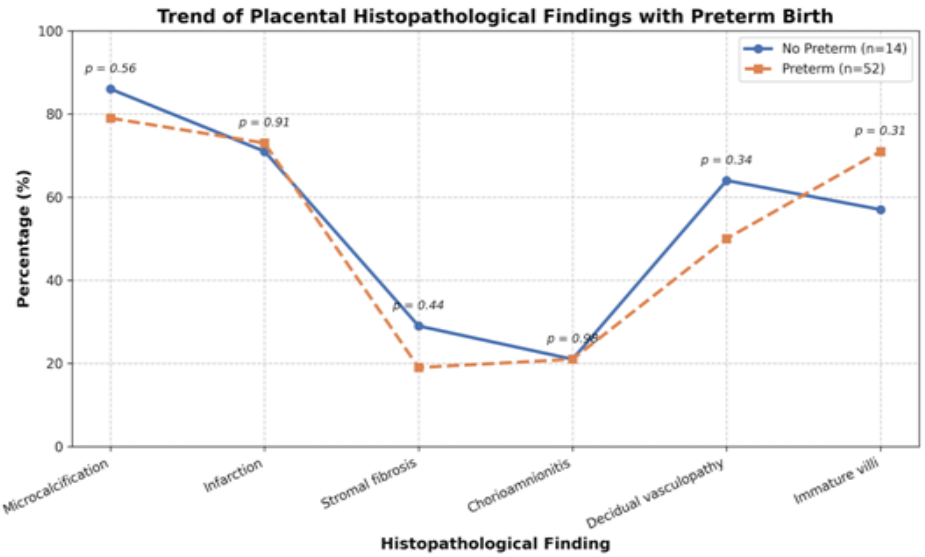

Table 8: Association of placental Histopathological findings with low birth weight

Histopathological finding	No LBW (n=32)	LBW (n=34)	p-value
Micro calcification	26 (81%)	27 (79%)	0.85
Infarction	25 (78%)	23 (68%)	0.33
Stromal fibrosis	6 (19%)	8 (24%)	0.63
Chorioamnionitis	8 (25%)	6 (18%)	0.46
Decidual vasculopathy	16 (50%)	19 (56%)	0.63
Immature villi	23 (72%)	22 (65%)	0.53

Observation: None of the placental Histopathological changes demonstrated a statistically significant association with low birth weight.

Table 9: Summary of Histopathological changes according to diagnosis

Histopathological finding	More common in	Statistical significance
Micro calcification	Eclampsia	No (p=0.12)
Infarction	Eclampsia	No (p=0.58)
Stromal fibrosis	Eclampsia	No (p=0.22)
Chorioamnionitis	Pre-eclampsia	No (p=0.54)
Decidual vasculopathy	Eclampsia	No (p=0.21)
Immature villi	Eclampsia	No (p=0.42)

Observation: Most placental lesions were more frequent in eclampsia than pre-eclampsia, except chorioamnionitis. However, none of these differences reached statistical significance.

Discussion

Hypertensive disorders of pregnancy are associated with significant maternal and fetal complications, and the placenta represents the primary organ affected by the pathological processes underlying these conditions. The present prospective observational study evaluated placental Histopathological changes among 66 women with hypertensive disorders of pregnancy, including 33 cases of pre-eclampsia and 33 cases of eclampsia.

In the present study, the mean maternal age was comparable between the eclampsia and pre-eclampsia groups (25.3 ± 5.3 years and 26.1 ± 5.1 years, respectively), with the majority of patients belonging to the 19–25 years age group. Similar age distributions have been reported in previous studies, where hypertensive disorders of pregnancy were more frequently observed among younger maternal age groups.[1,9] Younger maternal age has been identified as an important epidemiological factor associated with increased risk of hypertensive pregnancy disorders, although the relationship may vary depending on parity and socioeconomic factors.

The mean placental weight was 397.0 ± 62.3 g in the eclampsia group and 399.7 ± 125.5 g in the pre-eclampsia group. The fetoplacental ratio was also comparable between both groups. Reduced placental weight is commonly reported in severe hypertensive disorders due to impaired placental perfusion and increased ischemic injury. However, in the present study, no significant difference was observed between

the two groups. Similar findings have been reported by other researchers, suggesting that placental weight alone may not reliably differentiate between pre-eclampsia and eclampsia.[10]

Micro calcification was the most frequent Histopathological lesion observed in the present study, occurring in 80% of placentas, followed by infarction (73%) and immature villi (68%). Placental micro calcification is considered a marker of placental aging and chronic ischemic injury. Increased calcium deposition has been reported in pregnancies complicated by hypertension, reflecting vascular compromise and altered placental metabolism. Panday et al. reported placental micro calcification in a high proportion of women with pregnancy - induced hypertension, supporting the association between hypertensive disorders and calcific placental changes.[11]

Placental infarction was observed in 73% of cases in the present study. Infarction results from reduced maternal uteroplacental blood flow and is commonly associated with maternal vascular malperfusion lesions. The frequency of infarction was slightly higher among eclampsia patients (76%) compared with pre-eclampsia patients (70%), although the difference was not statistically significant. Similar observations have been reported in previous studies, where infarction was more frequent in severe hypertensive disease but showed considerable overlap between pre-eclampsia and eclampsia.[5], [12]

Decidual vasculopathy was observed in 53% of cases, with a higher frequency in eclampsia (61%) compared with pre-eclampsia (45%). Although the difference was statistically insignificant, the increased occurrence among eclampsia patients supports the role of severe maternal vascular dysfunction in advanced hypertensive disease. Decidual vasculopathy is considered one of the most specific placental lesions associated with pre-eclampsia and reflects defective spiral artery remodeling and maternal endothelial injury.[3],[6]

Stromal fibrosis was observed in 21% of cases and was more common among eclampsia patients. Fibrotic changes in chorionic villous stroma are associated with chronic hypoxia and prolonged placental injury.

Similarly, immature villi were identified in 68% of cases, with a higher frequency in eclampsia. Persistent immature villous development may indicate abnormal placental maturation and impaired adaptation to maternal vascular insufficiency.[4]

Acute chorioamnionitis was observed in 21% of placentas and was slightly more frequent in the pre-eclampsia group. Although inflammation has been implicated in the pathogenesis of hypertensive disorders, infectious and inflammatory lesions may occur independently of maternal hypertension. The lack of significant association in the present study suggests that inflammatory lesions may represent additional pathological processes rather than direct consequences of hypertensive disease.

The present study also evaluated associations between placental lesions and neonatal outcomes. Although micro calcification, infarction, and immature villi were commonly observed among preterm deliveries, none showed statistically significant associations. Similarly, no significant relationship was identified between placental Histopathological findings and low birth weight. These findings may be related to the relatively small sample size and the multi factorial nature of neonatal outcomes, which are influenced by gestational age, severity of maternal disease, and fetal factors.

Overall, the present study demonstrated that placental lesions associated with maternal vascular malperfusion were frequent among women with pre-eclampsia and eclampsia. Most lesions, including micro calcification, infarction, stromal fibrosis, decidual vasculopathy, and immature villi, showed a higher frequency among eclampsia patients, suggesting greater placental injury with disease severity. However, statistical significance was not achieved, possibly due to limited sample size.

Placental Histopathological examination remains an important investigative tool for understanding the pathological basis of hypertensive disorders of pregnancy. Recognition of these lesions may improve understanding of disease mechanisms and help correlate placental findings with maternal and neonatal outcomes.

Conclusion

The present study demonstrates that pre-eclampsia and eclampsia are associated with significant placental Histopathological alterations, predominantly micro calcification, infarction, immature villi, and decidual vasculopathy. Although these lesions were more frequently observed in eclampsia compared with pre-eclampsia, the differences were not statistically significant. Placental examination provides valuable insight into the effects of maternal hypertensive disorders on placental structure and may contribute to better understanding of disease severity and fetal outcomes. Further studies with larger sample sizes are required to establish stronger correlations between placental lesions, maternal disease severity, and neonatal outcomes.

References

1. American College of Obstetricians and Gynecologists. Gestational Hyper tension and Pree clampsia: ACOG Practice Bulletin No. 222. Obstet Gynecol. 2020; 135(6):e237-e260.

2. Steegers EAP, von Dadelszen P, Duvekot JJ, Pijnenborg R. Pre-eclampsia. Lancet. 2010; 376 (9741): 631-644.

3. Roberts JM, Hubel CA. The two stage model of preeclampsia: variations on the theme. Placenta. 2009; 30 (Suppl A):S32-S37.

4. Khong TY, Mooney EE, Ariel I, et al. Sampling and definitions of placental lesions: Amsterdam Placental Workshop Group Consensus Statement. Arch Pathol Lab Med. 2016; 140(7):698-713.

5. Burton GJ, Woods AW, Jauniaux E, Kingdom JCP. Rheological and physiological consequences of conversion of the maternal spiral arteries for uteroplacental blood flow during human pregnancy. Placenta. 2009; 30(6):473-482.

6. Brosens I, Pijnenborg R, Vercruysse L, Romero R. The "Great Obstetrical Syndromes" are associated with disorders of deep placentation. Am J Obstet Gynecol. 2011; 204(3):193-201.

7. Ogge G, Chaiworapongsa T, Romero R, et al. Placental lesions associated with maternal under perfusion are more frequent in early-onset than late-onset preeclampsia. J Perinat Med. 2011; 39(6):641-652.

8. Egbor M, Ansari T, Morris N, et al. Morpho metric placental changes in hypertensive disorders of pregnancy. Placenta. 2006; 27(9-10):959-964.

9. Duckitt K, Harrington D. Risk factors for pre-eclampsia at antenatal booking: systematic review of controlled studies. BMJ. 2005; 330:565.

10. Salafia CM, Zhang J, Miller RK, et al. Placental growth patterns affect birth weight for given placental weight. Birth. 2008; 35(2):87-94.

11. Panday K, Kaur S, Jain V. Placental Histopathological changes in pregnancy-induced hyper tension. J Clin Diagn Res. 2015;9(7):EC01-EC04.

12. Sankar KD, Bhanu PS, Ramakrishna BA, et al. Vascular changes in placenta in pregnancy induced hypertension. J Clin Diagn Res. 2013; 7 (10):2140-2144.