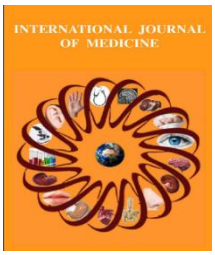

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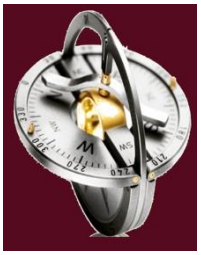

Original Research

A STUDY ON HISTOPATHOLOGICAL CHANGES IN THE PLACENTA IN PREECLAMPSIA.

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Abstract

Background: Preeclampsia is a multisystem hypertensive disorder of pregnancy that remains a major cause of maternal and perinatal morbidity and mortality worldwide. The placenta plays a pivotal role in the pathogenesis of preeclampsia, with abnormal placentation and uteroplacental insufficiency contributing to adverse maternal and fetal outcomes. Histopathological examination of placental tissue provides valuable insights into the underlying pathological processes and their association with disease severity and neonatal outcomes. *Aim:* To study the histopathological changes in the placenta in pregnancies complicated by preeclampsia and compare them with placentas from normotensive pregnancies.

Objectives:

1. To evaluate and compare the gross and microscopic histopathological features of placentas from preeclamptic and normotensive pregnancies.
2. To determine the association of placental histopathological changes with the severity of preeclampsia and selected fetal outcomes.

Materials and Methods: A hospital-based prospective comparative cross-sectional study was conducted over a period of 18 months in the Departments of Pathology and Obstetrics and Gynaecology of a tertiary care teaching hospital. The study included 100 placentas, comprising 50 placentas from women diagnosed with preeclampsia and 50 placentas from normotensive pregnant women. Gross placental examination included assessment of placental weight, dimensions, infarction, calcification, and cord abnormalities. Histopathological examination was performed on hematoxylin and eosin-stained sections to identify microscopic placental lesions. Maternal, obstetric, and fetal outcome parameters were recorded and statistically analyzed using Student's t-test, Chi-square test, and Fisher's exact test. A p-value of less than 0.05 was considered statistically significant. *Results:* The mean gestational age at delivery was significantly lower among women with preeclampsia than among normotensive controls (35.1 ± 2.6 weeks versus 38.4 ± 1.2 weeks, $p < 0.001$). Placental weight and dimensions were significantly reduced in the preeclampsia group ($p < 0.001$). Gross placental infarction and calcification were significantly more frequent among preeclamptic placentas. Histopathological findings such as increased syncytial knots (76%), villous infarction (68%), fibrinoid necrosis (62%), intervillous fibrin deposition (58%), decidual vasculopathy (44%), and accelerated villous maturation (54%) were significantly associated with preeclampsia ($p < 0.001$). Adverse fetal outcomes, including low birth weight (64%), fetal growth restriction (48%), preterm birth (56%), and NICU admission (44%), were significantly more common in pregnancies complicated by preeclampsia. Severe preeclampsia demonstrated a greater burden of placental pathology and poorer perinatal outcomes. *Conclusion:* Preeclampsia is associated with significant placental histopathological alterations that reflect uteroplacental vascular compromise and contribute substantially to adverse fetal outcomes. Histopathological examination of the placenta provides valuable clinicopathological information regarding disease severity and fetal prognosis. Routine placental evaluation in preeclamptic pregnancies can facilitate better understanding of disease mechanisms and improve maternal and neonatal management.

Keywords: *Preeclampsia; Placenta; Histopathology; Syncytial knots; Placental infarction; Fetal growth restriction; Maternal vascular malperfusion.*

Introduction

Preeclampsia is a multisystem hypertensive disorder of pregnancy characterized by the development of hypertension and proteinuria or end-organ dysfunction after 20 weeks of gestation in a previously normotensive woman. It remains one of the leading causes of maternal and perinatal morbidity and mortality worldwide, accounting for a significant proportion of adverse pregnancy outcomes. The disorder affects approximately 2%-8% of pregnancies globally and poses substantial challenges to maternal and fetal health, particularly in low- and middle-income countries. Placental dysfunction is considered the hallmark of preeclampsia and plays a central role in its pathogenesis, making histopathological evaluation of the placenta an indispensable tool for understanding the disease process. [1]

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Globally, hypertensive disorders of pregnancy contribute to nearly 14% of maternal deaths and are associated with complications such as placental abruption, intrauterine growth restriction (IUGR), preterm birth, fetal distress, and stillbirth. Inadequate trophoblastic invasion of maternal spiral arteries results in abnormal placentation, reduced uteroplacental blood flow, and chronic placental ischemia. These pathophysiological alterations trigger oxidative stress, endothelial dysfunction, and inflammatory responses that manifest as characteristic histopathological changes within the placenta. [2] Understanding these placental alterations provides valuable insights into fetal compromise and maternal disease severity.

The placenta serves as the primary interface between maternal and fetal circulation and is responsible for fetal nutrition, gas exchange, endocrine function, and immunological protection. In preeclampsia, placental morphology undergoes significant microscopic and macroscopic changes, including infarction, increased syncytial knots, fibrinoid necrosis, calcification, villous stromal fibrosis, intervillous fibrin deposition, and accelerated villous maturation. These pathological findings reflect chronic uteroplacental insufficiency and have been strongly associated with poor perinatal outcomes. Histopathological examination therefore plays an important role in establishing clinicopathological correlations in pregnancies complicated by preeclampsia. [3]

The prevalence of preeclampsia is increasing globally due to delayed childbearing, obesity, diabetes mellitus, and other metabolic disorders. According to the World Health Organization, hypertensive disorders complicate nearly one in ten pregnancies worldwide and disproportionately affect developing nations because of limited antenatal surveillance and delayed referral systems. Placental examination not only contributes to understanding disease mechanisms but also facilitates the identification of pregnancies at increased risk of recurrence and adverse neonatal outcomes. [4]

In India, hypertensive disorders of pregnancy constitute one of the major contributors to maternal mortality despite considerable advances in obstetric care. Preeclampsia affects approximately 5%–15% of pregnancies in India and remains a significant public health concern owing to its association with maternal complications such as eclampsia, HELLP syndrome, disseminated intravascular coagulation, and acute kidney injury. Indian studies have consistently demonstrated significant placental abnormalities in pregnancies complicated by preeclampsia, highlighting the role of placental pathology in determining fetal growth and neonatal survival. [5] Early recognition of placental changes can therefore aid clinicians in predicting pregnancy outcomes and optimizing antenatal management.

Histopathological examination of placental tissue provides a unique opportunity to evaluate the severity of uteroplacental vascular compromise. Several studies have reported a significant increase in placental infarction, syncytial knot formation, fibrinoid necrosis, and reduced placental weight among women with preeclampsia when compared with normotensive pregnancies. These morphological alterations have been directly associated with fetal growth restriction, low birth weight, and neonatal intensive care admissions. [6] Consequently, placental pathology has emerged as an important adjunct in understanding both maternal and fetal consequences of hypertensive disorders during pregnancy.

The clinicopathological evaluation of placental specimens contributes significantly to contemporary obstetric practice by providing evidence of placental insufficiency and identifying histopathological predictors of adverse outcomes. Detailed histological examination can also improve our understanding of disease severity and assist in counselling patients regarding future pregnancies. Furthermore, studies evaluating placental histopathological changes among Indian women are limited, necessitating further research in this area. [7] Therefore, the present study was undertaken to evaluate the histopathological changes occurring in the placenta of women with preeclampsia and to correlate these findings with the pathological processes associated with this important obstetric disorder.

AIM
To study the histopathological changes in placentas obtained from pregnancies complicated by preeclampsia and compare them with placentas from normotensive pregnancies.

OBJECTIVES
1. To evaluate and compare the gross and microscopic histopathological features of placentas from preeclamptic and normotensive pregnancies.
2. To determine the association of placental histopathological changes with the severity of preeclampsia and selected fetal outcomes, including gestational age at delivery, birth weight and fetal growth restriction.

Materials and Methods
Study design
A hospital-based, prospective comparative cross-sectional study was conducted.

Study setting
The study was conducted in the Department of Pathology in collaboration with the Department of Obstetrics and Gynaecology at Basaveshwara Medical College and Hospital, Chitradurga, Karnataka.

Study population
Placentas obtained after delivery from women with preeclampsia and normotensive pregnant women were included in the study.

Study groups
The participants were divided into two groups:

- **Case group:** 50 placentas from women diagnosed with preeclampsia.
- **Control group:** 50 placentas from normotensive women with singleton pregnancies.

Thus, the final sample comprised 100 placentas: 50 cases and 50 controls.

Sampling technique
Consecutive sampling was used. All eligible placentas received during the study period were included until the required sample size was achieved.

Definition of preeclampsia
Preeclampsia was diagnosed in a previously normotensive woman when new-onset hypertension developed after 20 weeks of gestation, with systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg on two measurements, accompanied by proteinuria or features of maternal organ dysfunction. Severe-range hypertension was defined as systolic blood pressure ≥160 mmHg or diastolic blood pressure ≥110 mmHg.

Inclusion criteria
Case group
1. Women with singleton pregnancies diagnosed with preeclampsia after 20 weeks of gestation.
2. Women who delivered in the study hospital during the study period.
3. Placentas received in an adequately preserved condition.
4. Participants who provided written informed consent.

Control group

1. Normotensive women with uncomplicated singleton pregnancies.
2. Women without proteinuria or clinical evidence of maternal systemic disease.
3. Women matched as far as possible for maternal age and gestational age.
4. Placentas received in an adequately preserved condition.

Exclusion criteria

1. Multiple pregnancies.
2. Chronic hypertension diagnosed before pregnancy or before 20 weeks of gestation.
3. Gestational diabetes mellitus or pre-existing diabetes mellitus.
4. Chronic renal, hepatic, autoimmune or cardiovascular disease.
5. Placenta previa, placental abruption unrelated to preeclampsia or major congenital fetal anomalies.
6. Clinical or histological evidence of acute chorioamnionitis.
7. Macerated stillbirths where autolysis prevented reliable histopathological interpretation.
8. Inadequately fixed, fragmented or poorly preserved placental specimens.

Data collection

Maternal information was recorded using a structured proforma. The variables included maternal age, parity, gestational age, blood-pressure measurements, proteinuria, severity of preeclampsia, mode of delivery and relevant laboratory findings. Fetal and neonatal details included sex, birth weight, Apgar score, presence of fetal growth restriction, preterm delivery, neonatal intensive care unit admission and perinatal outcome.

Collection and gross examination of placenta

Immediately after delivery, the placenta was collected, labelled and transported to the Department of Pathology. The umbilical cord and membranes were examined before trimming.

The following gross parameters were recorded:

- Placental weight after trimming the cord and membranes
- Placental dimensions and thickness
- Shape and completeness
- Maternal and fetal surface appearance
- Number and site of cotyledons
- Umbilical-cord length, diameter, insertion and number of vessels
- Presence of infarction, calcification, retroplacental hematoma, intervillous thrombi or other gross lesions

The placenta was serially sectioned at approximately 1-2 cm intervals. The extent of visible infarction and other lesions was recorded.

Tissue sampling and processing

Representative tissue sections were taken from:

1. Two full-thickness sections from apparently normal placental parenchyma.
2. One section from the placental margin with membranes.
3. Two sections from the umbilical cord, including the fetal end and placental insertion.
4. Additional sections from infarcts, calcified areas, hematomas, thrombi or other visible lesions.

The tissues were fixed in 10% neutral-buffered formalin for 24-48 hours. They were processed routinely, embedded in paraffin wax and sectioned at a thickness of approximately 4-5 µm. The sections were stained with haematoxylin and eosin.

Histopathological evaluation

The slides were examined under light microscopy for the following features:

- Increased syncytial knot formation
- Villous infarction
- Intervillous and perivillous fibrin deposition
- Fibrinoid necrosis
- Villous stromal fibrosis
- Accelerated villous maturation
- Distal villous hypoplasia
- Calcification
- Chorangiosis
- Villous vascular congestion
- Thrombosis and obliterative changes in fetal vessels
- Decidual vasculopathy, including fibrinoid necrosis and acute atherosis

The microscopic findings were compared between preeclamptic and normotensive groups. The preeclampsia group was further categorized into cases with and without severe features for subgroup analysis.

Statistical analysis

Data were entered into Microsoft Excel and analyzed using SPSS version 26.0. Continuous variables such as maternal age, gestational age, placental weight and birth weight were expressed as mean ± standard deviation or median with interquartile range. The independent-samples Student's t-test was used to compare normally distributed continuous variables between the two groups. The Mann-Whitney U test was used for non-normally distributed continuous variables. Categorical variables were expressed as frequencies and percentages. The Pearson Chi-square test was used to compare categorical variables. Fisher's exact test was used when expected cell frequencies were less than five. The association between specific placental lesions and adverse fetal outcomes was expressed using odds ratios with 95% confidence intervals. Multivariable logistic regression could be used to identify independent histopathological predictors of adverse perinatal outcomes after adjusting for gestational age and severity of preeclampsia. A p-value of <0.05 was considered statistically significant.

Results

Table 1. Comparison of maternal and obstetric characteristics between the study groups

Variable	Preeclampsia group (n = 50)	Control group (n = 50)	p value
Maternal age, years, mean ± SD	26.8 ± 4.5	25.9 ± 4.2	0.304
Primigravida, n (%)	28 (56.0)	23 (46.0)	0.317
Multigravida, n (%)	22 (44.0)	27 (54.0)	
Gestational age at delivery, weeks, mean ± SD	35.1 ± 2.6	38.4 ± 1.2	<0.001
Preterm delivery, n (%)	28 (56.0)	8 (16.0)	<0.001
Caesarean delivery, n (%)	31 (62.0)	18 (36.0)	0.009
Severe preeclampsia, n (%)	30 (60.0)	–	–
Non-severe preeclampsia, n (%)	20 (40.0)	–	–

Interpretation

The mean maternal age was comparable between the preeclampsia and control groups, with no statistically significant difference (p = 0.304). However, the mean gestational age at delivery was significantly lower among women with preeclampsia than among normotensive controls (35.1 ± 2.6 versus 38.4 ± 1.2 weeks; p < 0.001). Preterm delivery and caesarean delivery were significantly more frequent in the preeclampsia group. Among the preeclamptic women, 60% had disease with severe features.

Table 2. Comparison of gross placental findings between the study groups

Gross placental parameter	Preeclampsia group (n = 50)	Control group (n = 50)	p value
Placental weight, g, mean ± SD	418 ± 72	512 ± 65	<0.001
Placental diameter, cm, mean ± SD	15.6 ± 2.1	17.4 ± 1.8	<0.001
Placental thickness, cm, mean ± SD	2.1 ± 0.5	2.5 ± 0.4	<0.001
Placental weight <10th percentile, n (%)	30 (60.0)	8 (16.0)	<0.001
Gross placental infarction, n (%)	32 (64.0)	7 (14.0)	<0.001
Increased calcification, n (%)	24 (48.0)	10 (20.0)	0.003
Retroplacental hematoma, n (%)	10 (20.0)	2 (4.0)	0.028
Abnormal umbilical-cord insertion, n (%)	8 (16.0)	3 (6.0)	0.200

Interpretation

Placentas from women with preeclampsia had significantly lower mean weight, diameter and thickness than placentas from normotensive pregnancies. Gross infarction was present in 64% of preeclamptic placentas compared with 14% of controls. Increased calcification and retroplacental hematoma were also significantly more frequent in the preeclampsia group. However, the difference in abnormal umbilical-cord insertion was not statistically significant.

Table 3. Comparison of microscopic placental changes between the study groups

Histopathological finding	Preeclampsia group (n = 50)	Control group (n = 50)	Odds ratio (95% CI)	p value*
Increased syncytial knots	38 (76.0)	12 (24.0)	10.03 (4.05-24.84)	<0.001
Villous infarction	34 (68.0)	8 (16.0)	11.16 (4.22-29.54)	<0.001
Fibrinoid necrosis	31 (62.0)	9 (18.0)	7.43 (2.92-18.91)	<0.001
Villous stromal fibrosis	26 (52.0)	7 (14.0)	6.65 (2.48-17.85)	<0.001
Increased intervillous fibrin deposition	29 (58.0)	11 (22.0)	4.90 (2.06-11.67)	<0.001
Decidual vasculopathy/acute atherosis	22 (44.0)	3 (6.0)	12.31 (3.39-44.72)	<0.001
Accelerated villous maturation	27 (54.0)	9 (18.0)	5.35 (2.16-13.25)	<0.001
Increased calcification	25 (50.0)	12 (24.0)	3.17 (1.36-7.39)	0.012
Chorangiosis	8 (16.0)	3 (6.0)	2.98 (0.74-12.01)	0.200

Interpretation

The frequencies of increased syncytial knots, villous infarction, fibrinoid necrosis, villous stromal fibrosis, intervillous fibrin deposition, decidual vasculopathy and accelerated villous maturation were significantly higher in placentas from preeclamptic pregnancies. Decidual vasculopathy showed one of the strongest associations with preeclampsia, with approximately 12 times higher odds than in controls. Chorangiosis was more frequent in the preeclampsia group, but the difference was not statistically significant.

Table 4. Comparison of fetal and neonatal outcomes between the study groups

Fetal/neonatal outcome	Preeclampsia group (n = 50)	Control group (n = 50)	Odds ratio (95% CI)	p value*
Birth weight, kg, mean ± SD	2.32 ± 0.58	3.02 ± 0.42	–	<0.001†
Low birth weight (<2.5 kg)	32 (64.0)	10 (20.0)	7.11 (2.91-17.37)	<0.001
Fetal growth restriction	24 (48.0)	5 (10.0)	8.31 (2.78-24.80)	<0.001
Preterm birth	28 (56.0)	8 (16.0)	6.68 (2.62-17.07)	<0.001
NICU admission	22 (44.0)	7 (14.0)	4.83 (1.83-12.71)	0.002
Five-minute Apgar score <7	15 (30.0)	4 (8.0)	4.93 (1.51-16.09)	0.009
Stillbirth	5 (10.0)	1 (2.0)	5.44 (0.61-48.32)	0.204

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nterpretation

The mean birth weight was significantly lower in the preeclampsia group than in the control group. Low birth weight, fetal growth restriction, preterm birth, NICU admission and a five-minute Apgar score below 7 were significantly more frequent among pregnancies complicated by preeclampsia. Stillbirth was numerically higher in the preeclampsia group, but the difference did not reach statistical significance, possibly because of the small number of events.

Table 5. Association between severity of preeclampsia, placental lesions and fetal growth restriction among cases

Variable	Severe preeclampsia (n = 30)	Non-severe preeclampsia (n = 20)	Odds ratio (95% CI)	p value*
Increased syncytial knots	27 (90.0)	11 (55.0)	7.36 (1.66-32.63)	0.007
Villous infarction	24 (80.0)	10 (50.0)	4.00 (1.11-14.35)	0.034
Decidual vasculopathy	18 (60.0)	4 (20.0)	6.00 (1.62-22.16)	0.008
Fibrinoid necrosis	22 (73.3)	9 (45.0)	3.36 (0.93-12.17)	0.073
Fetal growth restriction	19 (63.3)	5 (25.0)	5.18 (1.49-18.06)	0.010
Low birth weight	23 (76.7)	9 (45.0)	4.02 (1.14-14.12)	0.030
NICU admission	18 (60.0)	4 (20.0)	6.00 (1.62-22.16)	0.008

Interpretation
Severe preeclampsia was significantly associated with increased syncytial knots, villous infarction and decidual vasculopathy. Fetal growth restriction, low birth weight and NICU admission were also significantly more frequent among women with severe preeclampsia. Fibrinoid necrosis was more common in severe preeclampsia, but the association was not statistically significant at the 5% level.

Overall interpretation
The study findings indicated that preeclampsia was associated with reduced placental size and weight and a markedly increased frequency of placental ischemic and vascular lesions. Increased syncytial knots, villous infarction, fibrinoid necrosis, intervillous fibrin deposition and decidual vasculopathy were the principal histopathological abnormalities. These placental changes were more pronounced in severe preeclampsia and were accompanied by higher frequencies of fetal growth restriction, low birth weight, preterm delivery and NICU admission. The findings support a strong relationship between the severity of maternal hypertensive disease, placental insufficiency and adverse perinatal outcomes.

Discussion

The present study evaluated the histopathological alterations in placentas obtained from pregnancies complicated by preeclampsia and compared them with placentas from normotensive pregnancies. The findings demonstrated significant differences in maternal, placental, and fetal parameters, thereby emphasizing the role of placental pathology in the pathogenesis and adverse outcomes associated with preeclampsia. The mean gestational age at delivery in the preeclampsia group was significantly lower than that of the control group (35.1 ± 2.6 weeks versus 38.4 ± 1.2 weeks, $p < 0.001$), with preterm delivery observed in 56% of cases. Similar findings were reported by Majumdar et al., who observed significantly earlier deliveries and increased rates of preterm births among women with severe preeclampsia, highlighting the association between placental insufficiency and premature termination of pregnancy.[8] Furthermore, Kambale et al. demonstrated that severe placental vascular pathology is strongly associated with shortened gestational duration in hypertensive pregnancies. [9]

Gross placental examination revealed significantly reduced placental weight, diameter, and thickness among women with preeclampsia. The mean placental weight in the present study was 418 ± 72 g in cases compared to 512 ± 65 g among controls ($p < 0.001$). Udainia and Jain similarly reported a significantly reduced placental weight in preeclamptic pregnancies, with approximately 64% of placentas exhibiting gross infarction and increased calcification. [10] Reduced placental dimensions reflect impaired trophoblastic invasion and diminished uteroplacental blood flow, which subsequently compromise fetal nutrition and growth. Such gross morphological changes serve as valuable indicators of placental dysfunction and fetal compromise.

Placental infarction was observed in 68% of preeclamptic placentas in the present study compared with 16% among controls. Increased syncytial knot formation was the most frequent microscopic lesion, occurring in 76% of cases. These findings are comparable to those of Geetha et al., who reported placental infarction in nearly 70% and syncytial knots in over 75% of placentas obtained from women with preeclampsia. [11] Syncytial knot formation is considered a compensatory response to chronic uteroplacental hypoxia and is among the earliest histological markers of placental ischemia. Its increased frequency in preeclampsia reflects accelerated villous maturation resulting from chronic placental stress.

The present study demonstrated significantly increased frequencies of fibrinoid necrosis (62%), villous stromal fibrosis (52%), and intervillous fibrin deposition (58%) in the preeclampsia group. Similar observations have been documented by Tiwari and colleagues, who reported extensive fibrinoid necrosis and stromal fibrosis among placentas of women with severe hypertensive disorders of pregnancy. [12] These pathological alterations indicate progressive placental vascular injury and impaired maternal-fetal exchange. Excessive fibrin deposition within the intervillous spaces has been associated with reduced placental perfusion and an increased risk of fetal growth restriction.

Decidual vasculopathy and acute atherosis were identified in 44% of preeclamptic placentas in the present study and were significantly associated with severe disease ($p < 0.001$). Khong et al. emphasized that decidual vasculopathy represents the hallmark lesion of maternal vascular malperfusion and is highly predictive of adverse pregnancy outcomes, including severe preeclampsia and intrauterine growth restriction. [13] The presence of these vascular lesions reflects abnormal spiral artery remodelling and inadequate placental perfusion, which constitute the central pathogenic mechanisms underlying preeclampsia.

Accelerated villous maturation was observed in 54% of placentas in the present study and was significantly associated with preeclampsia. Similar findings have been reported by Ogge et al., who demonstrated that accelerated villous maturation and distal villous hypoplasia are common placental manifestations of maternal vascular malperfusion and are frequently encountered in hypertensive pregnancies. [14] These microscopic changes represent adaptive responses to placental hypoxia and contribute to impaired placental function.

Fetal outcomes were significantly compromised in pregnancies complicated by preeclampsia. The mean birth weight in the preeclampsia group was 2.32 ± 0.58 kg compared with 3.02 ± 0.42 kg among controls ($p < 0.001$). Low birth weight and fetal growth restriction were observed in 64% and 48% of cases, respectively. Similar findings have been documented by Salmani et al., who reported significantly lower birth weights and increased incidences of fetal growth restriction among women with preeclampsia owing to placental insufficiency. [15] The strong association between placental pathology and fetal growth impairment underscores the critical role of adequate placental vascularization in fetal development.

The present study also demonstrated significantly increased rates of NICU admission (44%), preterm birth (56%), and low Apgar scores among neonates born to preeclamptic mothers. Egbor et al. similarly reported increased neonatal morbidity among pregnancies complicated by placental ischemic lesions and maternal hypertension. [16] Placental abnormalities compromise oxygen and nutrient transfer to the foetus, resulting in neonatal complications that often necessitate intensive care management.
Among women with severe preeclampsia, syncytial knots (90%), villous infarction (80%), and decidual vasculopathy (60%) were significantly more frequent than among women with non-severe disease. These findings are consistent with those reported by Raymond et al., who observed that the severity of placental lesions parallels the clinical severity of maternal disease and predicts adverse perinatal outcomes. [17] Severe preeclampsia is therefore associated with more profound placental vascular compromise, explaining its poorer maternal and neonatal prognosis.

The present study further substantiates the concept that placental histopathology serves as a reliable indicator of disease severity and fetal outcome in pregnancies complicated by preeclampsia. Histopathological evaluation not only enhances our understanding of the underlying pathophysiological mechanisms but also facilitates clinicopathological correlation in hypertensive disorders of pregnancy. As demonstrated by Sebire and colleagues, systematic placental examination provides valuable prognostic information regarding maternal vascular malperfusion and fetal compromise and should be incorporated into routine pathological assessment whenever feasible. [18]

In summary, the findings of the present study establish that preeclampsia is associated with significant gross and microscopic placental abnormalities that correlate strongly with adverse fetal outcomes. Early identification and comprehensive histopathological examination of placental lesions can contribute substantially to improving maternal and neonatal care and may provide useful information for counselling women regarding subsequent pregnancies.

Conclusion

The present study demonstrated that preeclampsia is associated with significant gross and microscopic placental abnormalities resulting from uteroplacental vascular insufficiency. Placentas from preeclamptic pregnancies exhibited reduced placental weight and dimensions, along with characteristic histopathological changes such as increased syncytial knots, villous infarction, fibrinoid necrosis, intervillous fibrin deposition, decidual vasculopathy, and accelerated villous maturation. These placental lesions were more pronounced in severe preeclampsia and showed a significant association with adverse fetal outcomes, including low birth weight, fetal growth restriction, preterm delivery, and increased NICU admissions. Histopathological examination of the placenta serves as a valuable diagnostic and prognostic tool in understanding the pathogenesis and severity of preeclampsia. Routine placental evaluation in hypertensive pregnancies may aid in clinicopathological correlation, improve perinatal outcome assessment, and provide useful information for counseling women regarding future pregnancies.

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