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Original Research Article

Placental morphological and histopathological changes in preeclampsia and eclampsia: a prospective case-control study using an objective scoring system

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ABSTRACT

Background: The hypertensive disorders of pregnancy (HDP), particularly preeclampsia (PE) and eclampsia (EC) play a major role in maternal and perinatal morbidity. Despite extensive descriptive work on placental lesions in these conditions, only a few studies have attempted to objectively quantify the morphological damage. This study was undertaken to apply a structured histopathological scoring system to placental lesions in PE and EC and to determine whether the scores correlate with the severity of disease and adverse fetal outcome.
Methods: This prospective case-control study included 100 placentas: 50 from PE, 25 from EC, and 25 from normotensive pregnancies as controls. Gross and microscopic features were assessed using a semi-quantitative histopathological scoring system evaluating fibrin deposition, maternal floor infarction, syncytial knot, calcification, and villous basement membrane thickening. Scores were correlated with placental weight and fetal outcome, indicating uteroplacental insufficiency.
Results: Placenta; weight and thickness were significantly reduced in hypertensive cases compared with controls ($p<0.001$). Histological lesions such as fibrin deposition, maternal floor infarction, and syncytial knotting were markedly increased in the PE and EC groups ($p<0.001$). The median composite histopathological score was 4 in controls, 6 in PE, and 8 in EC. Higher scores were significantly associated with low placental weight and adverse fetal outcome.
Conclusions: Placental histopathological scoring offers an objective and reproducible approach to quantity morphological damage in PE and EC. The scoring system correlates with disease severity and fetal outcome, underscoring its potential value as a morphological marker of uteroplacental insufficiency.

Keywords: Hypertensive disorders of pregnancy, Placental pathology, Morphological scoring

INTRODUCTION

Hypertensive disorders of pregnancy (HDP) remain the leading cause of maternal and perinatal mortality and morbidity globally. They complicate approximately 4-8% of pregnancies and contribute to 10-15% of maternal deaths globally.[1] Among the various causes of HDP, PE and EC represent the most severe forms, and are characterized by hypertension and multisystem endothelial dysfunction, which results in abnormal placental perfusion resulting in uteroplacental insufficiency.[2] As placenta, is a critical interface between the fetus and the mother, it reflects characteristic pathological changes through structural and vascular alterations.[3] Examination of the placenta, therefore, provides valuable insights into the underlying uteroplacental insufficiency and its relationship with maternal and fetal outcomes.[4]

Numerous studies have documented the histopathological spectrum of placental changes in PE and EC, including

fibrinoid necrosis, villous infarction, syncytial knotting, and villous basement membrane thickening.[3,4] However, most prior studies have been descriptive, limiting reproductivity and comparison between investigators. Quantitative or semi-quantitative systems that objectively grade these lesions are scarce. Establishing a standardized scoring method could help to objectively correlate the morphological damage with clinical disease severity and perinatal outcome.[5,6]

In light of these limitations, the present study was undertaken, which applied a composite histopathological scoring system incorporating five key parameters: fibrin deposition, maternal floor infarction, syncytial knotting, calcification, and villous basement membrane thickening. Each lesion was graded on a defined scale to generate a comprehensive score reflecting the degree of placental injury. Such a systematic assessment may serve as a morphological correlate to maternal uteroplacental insufficiency and provide a reliable indicator of disease severity.[8]

The present study was undertaken to evaluate the morphological changes in the placenta from pregnancies complicated by PE and EC using an objective histopathological score with placenta weight and perinatal outcome to assess the degree of maternal uteroplacental insufficiency.

METHODS

Study design and setting

The present prospective case-control study was conducted in the department of pathology in collaboration with the department of obstetrics and gynaecology, Moti Lal Nehru medical college, Prayagraj, Uttar Pradesh, over a period of 24 months (June 2019 to June 2021. Ethical approval for the study was obtained from institutional ethics committee. Written informed consent was taken from all participants.

Study population and sample size

A total of 100 pregnant women were included in the study, of whom 75 had hypertensive disorder of pregnancy and 25 were normotensive controls. The hypertensive group consisted of 50 women with PE and 25 with EC.

Sample size was determined based on previously published data demonstrating a mean difference of 58 grams in placental weight between hypertensive and normotensive pregnancies. Assuming a standard deviation of 90 gm, 80% power, and 95% CI, minimum sample size required was calculated as 42 per group; hence, 100 placentas were included to improve precision.[9]

Inclusion criteria

Women aged >18 years and gestational age >24 weeks, diagnosed as normotensive or hypertensive according to the American college of obstetricians and gynecologists (ACOG) 2014 guidelines.

Gestation hypertension: Blood pressure ≥140/90 mmHg after 20 weeks of gestation without proteinuria.

PE: Blood pressure ≥140/90 mmHg with proteinuria (> 300 mg/day or ≥1 g/l [2+] on dipstick).

EC: PE complicated by new onset seizures or coma, not attributable to other causes.

Exclusion criteria

Women with chronic hypertension, diabetes mellitus, hepatitis B and C, HIV, and COVID were excluded.

Clinical data collection

Clinical details, including maternal age, gestation age at delivery, parity, and blood pressure at admission, were recorded. Systolic and diastolic blood pressure and urine albumin levels were measured by the dipstick method. The mode of delivery was noted. Neonatal outcomes, including birth weight, stillbirth, and fetal death, were recorded.

For this study, the following definitions were used in accordance with standard obstetric guidelines:[10]

Low birth weight (LBW): Neonates weighing less than 2.5 kilograms (2,500 grams) at birth, irrespective of gestational age.

Stillbirth: Fetal death occurring at or after 28 weeks of gestation, defined as delivery of a baby with no signs of life (no breathing, heartbeat, or umbilical cord pulsation).

Normal fetal outcome: Liveborn infants with birth weight ≥2.5 kg and no congenital or obstetric complications.

Birth weights were obtained using standardized electronic scales within one hour of delivery.

Specimen collection and gross examination

Immediately following delivery, the placenta, along with attached membranes and umbilical cord, was collected, washed in running tap water, and fixed in 10% neutral buffered formalin.

Gross examination included recording placental weight and thickness. Lesions such as infarction, calcification, and retroplacental hematoma were recorded. Representative tissue sections were obtained from the cord, membranes, and at least three areas of the placental disc, including any visible lesions.

For this study, following definitions were applied based on standard obstetric and placental pathology references.[7,11]

Low placental weight

Placental weight less than 400 grams after trimming membranes and cord. This threshold has been used in previous studies as an indicator of maternal uteroplacental insufficiency.

Normal placental weight

Placental weight ≥400 grams after trimming.

Reduced placental thickness

Placental parenchymal thickness less than 2.0 cm measured at the center of the disc after fixation in 10% formalin.

Normal placental thickness

Thickness ≥2.0 cm, measured similarly.

Placental weight and thickness were measured immediately after delivery using standardized protocols before fixation.

Histopathological evaluation

Paraffin-embedded tissue was sectioned at 4-5 µm thickness and stained with hematoxylin and eosin (H and E). Each section was evaluated for five histopathological features: Fibrin deposition, maternal floor infarction, syncytial knotting, calcification and villous basement membrane thickening.

Each parameter was scored semi-quantitatively.[7]

0=Absent, 1=mild/focal involvement and 2=moderate to severe involvement or diffuse distribution.

For syncytial knots, presence or absence (0-1) was recorded. The cumulative histopathological score ranged from 0 to 9, where higher scores indicated greater placental injury.

Statistical analysis

All data were entered in Microsoft excel and analyzed using SPSS version. Continuous variables were expressed as mean±standard deviation (SD) and compared using the student's t test or ANOVA. Categorical variables were compared using the Chi-square test. A $p<0.05$ was considered statistically significant.

RESULTS

This study was conducted in the department of pathology, in collaboration with the department of obstetrics and gynaecology, Moti Lal Nehru medical college, Prayagraj, over a period of two years. A total of 100 placentas were examined, of which 50 were from PE patients, 25 from eclamptic patients, and 25 from normotensive pregnancies serving as controls.

Clinical profile

Table 1 presents the demographic and clinical characteristics of the study groups. The majority of the women in all groups were aged 21-25 years with no statistically significant difference between the groups (p=0.816).

In terms of gestational age, preterm deliveries (30-35 weeks) were more frequent among PE (56%) and EC (40%) women compared to controls (20%), and this difference was statistically significant (p=0.001).

Most PE (48%) and EC (56%) patients were primigravidae, but this difference was statistically significant (p=0.066). The majority of cases in both groups were delivered by caesarean section (PE=90%; EC=96%) compared to 60% in controls (p=0.005).

Mean systolic and diastolic blood pressures were significantly higher in the case groups compared to controls. Proteinuria was present in all PE and EC cases but absent in the control group.

Fetal outcome

Table 2 summarizes fetal outcomes. The incidence of low birth weight was higher in PE (40%) and EC (32%) compared to normal pregnancies (NP) (28%), though not statistically significant (p=0.596). Stillbirths were significantly more frequent among cases (PE=20%; EC=36%) than controls (16%) (p=0.001).

Gross morphology of placenta

Table 3 shows the gross placental findings. Placentas from hypertensive pregnancies were lighter and thinner than those from normotensive pregnancies. Low placental weight (<400 g) was found in 56% of PE and 68% of EC cases versus 36% of controls (p=0.0024).

Placental thickness <2 cm was observed in 52% and 64% of PE and EC groups, respectively, compared to 36% of controls. (p=0.005).

Infarction was the most frequent gross abnormality, present in 52% of PE and 60% of EC cases compared to 28% of controls (p=0.001) (Figure 1). Calcification and retroplacental hematoma (Figure 2) were observed in all groups without a significant difference (p>0.05) (Figure 1).

Microscopic (Histopathological) findings

Table 4 outlines the histopathological findings. Infarction (Figure 2 A), calcification (Figure 2 B), syncytial knotting

(Figure 2 C), and villous basement membrane thickening (Figure 2 D) were more frequent in hypertensive cases compared to controls.

Significant histopathological differences were noted for infarction, syncytial knotting and basement membrane thickening between case and control groups (p<0.05).

Objective histopathological scoring

Each placenta was assigned a composite histopathological score based on the semi-quantitative system described by Donthi et al.[7] Table 5 demonstrates that higher scores were observed in PE and EC groups. Significant differences were observed in fibrin deposition, maternal floor infarction, and syncytial knotting (p<0.05), while calcification and villous basement membrane thickening showed no significant association.

Median objective scores (IQR) were 4 for normal pregnancies, 6 for PE, and 8 for EC, indicating progressively severe placental pathology across disease severity as shown in Table 6. Although the numerical rise in the score paralleled disease severity, the difference did not reach statistical significance (p=0.925).

These findings indicate that with increasing clinical severity of hypertensive disorders, there is a corresponding escalation in histopathological damage within the placenta.

Table 1: Clinical characteristics of PE, EC and NP groups.

Characteristics	NP, n=25 (%)	PE, n=50 (%)	EC, n=25 (%)	P value	Statistical significance
Age (in years)					
<20	3 (11)	8 (16)	4 (16)	0.816	Not significant
21-25	14 (57)	22 (45)	12 (48)		
26-30	6 (22)	15 (27)	6 (22)		
31-35	2 (10)	5 (12)	3 (14)		
Gestational age (in weeks)					
25-30	2 (8)	7 (14)	7 (28)	0.001	Significant
30-35	4 (16)	28 (56)	10 (40)		
35-40	19 (76)	15 (30)	8 (32)		
Gravida					
Multigravida	14 (56)	26 (52)	11 (44)	0.066	Not significant
Primigravida	11 (44)	24 (48)	14 (56)		
Mode of delivery					
Induced vaginal delivery	10 (40)	5 (1)	1 (4)	0.005	Significant
Caesarean section	15 (60)	45 (90)	24 (96)		
Systolic BP (mmHg)	118.88±7.57	148.28±5.406	178±8.98	0.001	Significant
Diastolic BP (mmHg)	69.39±18.99	87.96±12.83	98.48±3.52	0.001	Significant
Proteinuria	-	+	++		

Table 2: Fetal outcomes among study groups.

Variables	Normal, n=25 (%)	PE, n=50 (%)	EC, n=25 (%)	P value	Statistical significance
Normal outcome*	13 (52)	18 (36)	8 (32)		
Low birth weight **	7 (28)	20 (40)	8 (32)	0.596	Not significant
Stillbirth [#]	4 (16)	12 (20)	9 (36)	0.001	Significant

Normal outcome-Healthy baby weighing >2.5 kg, free of any congenital anomaly or obstetric damage; low birth weight** Baby weighing <2.5 kg; Still birth[#]– baby born with no signs of life at 28 weeks of pregnancy or more.

Table 3: Gross placental morphology in cases (PE and EC) and control (NP) groups.

Pathological features of placenta	NP, n=25 (%)	PE, n=50 (%)	EC, n=25 (%)	P value	Statistical significance
Trimmed weight of placenta					
<400 gm	9 (36)	28 (56)	17 (68)	0.0024	Significant
>400 gm	16 (64)	22 (44)	8 (32)		
Placental thickness					
<2 cm	9 (36)	26 (52)	16 (64)	0.005	Significant
>2 cm	16 (64)	24 (48)	8 (32)		

Continued.

Pathological features of placenta	NP, n=25 (%)	PE, n=50 (%)	EC, n=25 (%)	P value	Statistical significance
Infarction					
Present	7 (28)	26 (52)	15 (60)	0.001	Significant
Absent	18 (72)	24 (48)	10 (40)		
Calcification					
Present	12 (48)	17 (34)	11 (44)	0.118	Not significant
Absent	13 (52)	33 (66)	14 (56)		
Retroplacental hematoma					
Present	12 (40)	16 (32)	12 (48)	0.607	Not significant
Absent	13 (52)	34 (68)	13 (52)		

Table 4: Distribution of major histo-morphological features in cases (PE and EC) and control (NP) groups.

Histological features	NP, n=25 (%)	PE, n=50 (%)	EC, n=25 (%)	P value	Statistical significance
Infarction					
Present	4 (16)	28 (56)	13 (52)	0.004	Significant
Absent	21 (84)	22 (44)	12 (48)		
Calcification					
Present	8 (32)	14 (28)	12 (48)	0.007	Significant
Absent	17 (68)	36 (72)	13 (52)		
Fibrinoid necrosis					
Present	10 (40)	24(48)	14 (56)	0.731	Not significant
Absent	15 (60)	25 (50)	11 (44)		
Fibrin deposition					
Present	15 (60)	25 (50)	10 (40)	0.619	Not significant
Absent	10 (40)	24 (50)	15 (60)		
Syncytial knotting					
Present	14 (56)	35 (70)	20 (80)	0.011	Significant
Absent	11 (44)	15 (30)	5 (20)		
Villous BM thickening					
Present	10 (40)	22 (44)	14 (56)	0.011	Significant
Absent	15 (60)	28 (56)	11 (44)		

Table 5: Distribution of placental histo-morphological features using the objective scoring system.

Histological features	Criteria	NP, n=25 (%)	PE, n=50 (%)	EC, n=25 (%)	P value	Statistical significance
Fibrin deposition						
0	Absent	15 (60)	6 (12)	2 (8)	<0.0001	Significant
1	Intravillous/perivillous	9 (40)	32 (64)	14 (56)		
2	Intravillous + perivillous	1 (4)	12 (24)	9 (36)		
Maternal floor infarction						
0	Absent	14 (56)	3 (12)	2 (8)	0.0001	Significant
1	Single area	6 (24)	30 (60)	13 (52)		
2	Multiple area	5 (20)	17 (34)	10 (40)		
Calcification						
0	Absent	12 (48)	17 (34)	12 (48)	0.2308	Not significant
1	Focal	8 (32)	19 (38)	5 (20)		
2	Diffuse	5 (20)	14 (28)	8 (32)		
Syncytial knots						
0	Absent	8 (32)	8 (16)	5 (20)	0.019	Significant
1	Present	17 (68)	42 (84)	20 (80)		
Villous BM thickening						
0	Absent	12 (48)	23 (46)	11 (44)	0.825	Not significant
1	Thickened	12 (48)	24 (48)	12 (48)		
2	Hyalinized	1 (4)	3 (6)	2 (8)		

Table 6: Association of histopathological scores with clinical diagnosis.

Histopathological diagnosis	NP	PE	EC	P value
Median (IQR)	4 (2)	6 (1)	8 (3)	0.925

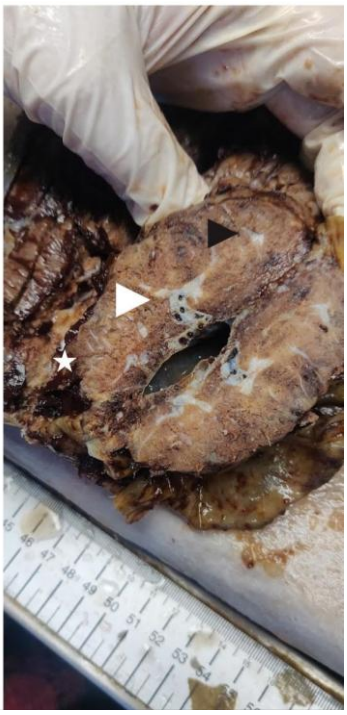

Figure 1: Gross morphology of placenta in HDP.

*Gross placental photograph showing multiple pathological findings in hypertensive pregnancy. Infarcted areas (white arrows), foci of calcification (black arrows), and a retroplacental hematoma (asterisk) are seen. These gross features indicate maternal vascular malperfusion consistent with PE/EC.

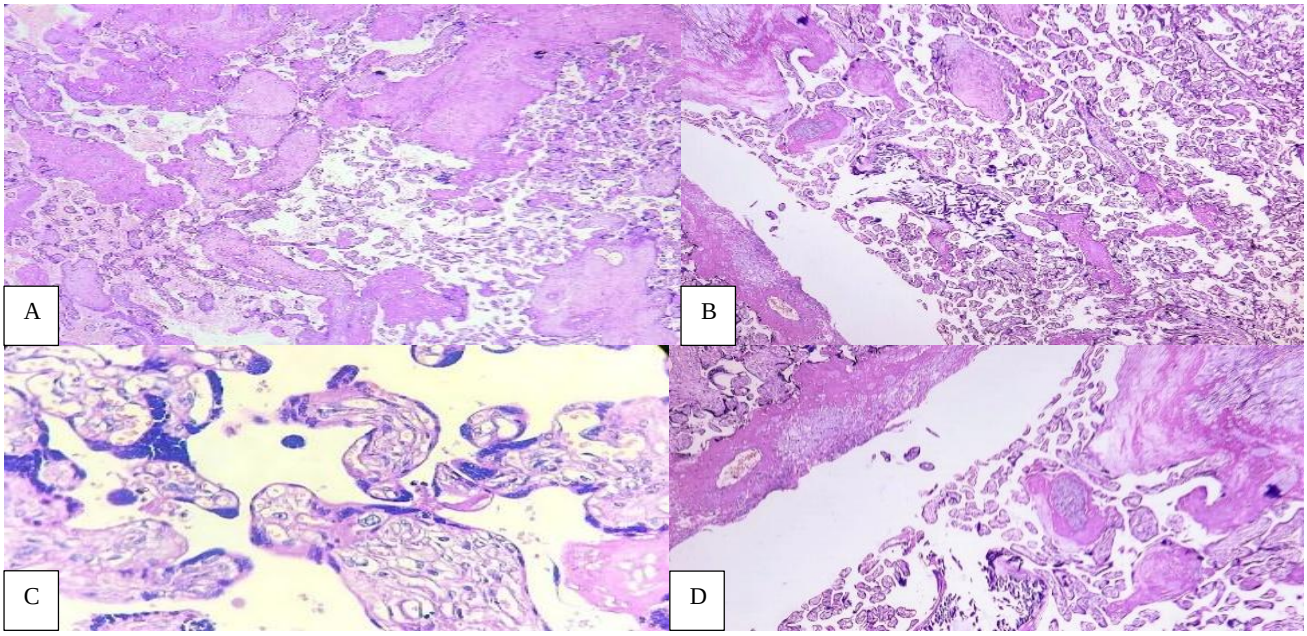

Figure 2 (A-D): Microscopic features of placenta in PE and EC.

*Representative microphotographs showing histopathological lesions observed in hypertensive placentas. (A) Villous infarction (H and E, 40×) showing necrotic villi with ghost outlines. (B) Focal calcification (H and E, 40×) seen as basophilic granular deposits. (C) Syncytial knotting (H and E, 40×) showing increased nuclear aggregates at villous peripheries. (D) Villous basement membrane thickening (H and E, 40×) demonstrating hyalinization of the villous stroma. All lesions indicate chronic uteroplacental insufficiency.

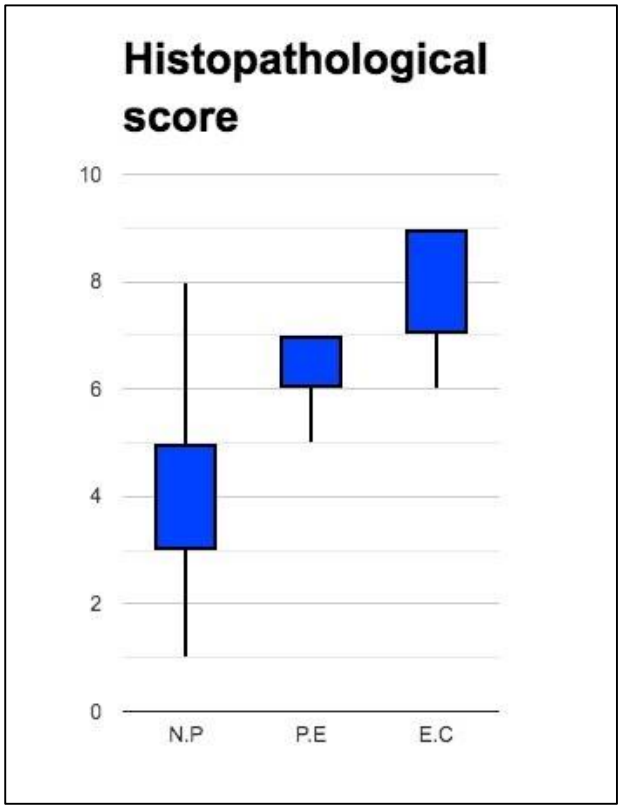

Figure 3: Comparative histopathological scores among normal, PE, and EC groups.

*Box-and-whisker plot showing median and interquartile range (IQR) of composite histopathological scores across study groups. The scores demonstrate a stepwise increase from normal pregnancies (median=4, IQR=2) to PE (median=6, IQR=1) and EC (median=8, IQR=3), reflecting progressive placental pathology severity.

DISCUSSION

The placenta has long been regarded as the mirror of perinatal health, reflecting the maternal and fetal environment throughout gestation. In HDP, particularly PE and EC, placental morphology provides a crucial link to understanding uteroplacental insufficiency and adverse perinatal outcomes. Several studies have demonstrated that structural and vascular alterations in the placenta correspond to disease severity and clinical prognosis.

The present study was undertaken to examine the gross and microscopic changes in placentas from pregnancies complicated by PE and EC, and to develop an objective semi-quantitative histopathological scoring system. This approach aimed to establish a uniform, reproducible method for reporting placental pathology and correlating morphological findings with clinical outcomes.

In the present study, most patients with PE and EC were in the 21-25 year age group, similar to findings of other studies.[12] The occurrence of PE and EC in younger women, particularly primigravidae, supports the immune maladaptation theory of maternal tolerance to paternal antigens, which leads to defective artery remodelling.[13,14]

In the current study, preterm delivery (30-35 weeks) was significantly higher in the cases group in comparison to the normotensive control, which is in concordance with other studies.[15,16] These results from uteroplacental insufficiency, which is secondary to maternal vascular malperfusion, which results in early onset of labor or serves as an indication for cesarean delivery in hypertensive pregnancies.[17]

A predominance of primigravida in the PE and EC group was observed, which is our study as described in other studies.[18] The underlying mechanism likely involves first-time maternal exposure to trophoblastic antigens, resulting in incomplete immunological tolerance.[19]

Regarding mode of delivery, the majority of PE and EC patients in our study underwent cesarean section (90% PE, 96% EC), comparable to findings of other studies, which reported a cesarean rate exceeding 80% among severe PE cases due to maternal or fetal indications such as uncontrolled hypertension or fetal distress.[20]

In this study, low birth weight was observed in 40% of PE and 32% of EC cases of neonates, with a threefold increase in stillbirth in comparison to the control group. Similar trends were reported by other studies.[20,21] Placental malperfusion and chronic fetal hypoxia lead to hypertensive pregnancies, which result in intrauterine growth restriction and perinatal mortality.[22] In an Indian cohort, Akhlaq et al reported that 35-45% of neonates born to mothers who had PE had low birth weight, mirroring our findings.[23]

Collectively, these results prove that maternal hypertension correlates strongly with adverse fetal outcomes. The increased cesarean rate and perinatal loss in our study reflect the obstetricians' attempt to optimize survival in the context of maternal uteroplacental insufficiency.[24]

In the present study, the mean placental weight was lower (<400 g) in the case group, i. e, PE and EC, in comparison to the control group. Similar observations were made by other authors as well, who demonstrated that hypertensive disorders lead to a 15-20% reduction in the placental mass.[15,23,25] The reduction in placental weight is due to maternal vascular malperfusion resulting in chronic uteroplacental insufficiency, which leads to ischemic damage, resulting in a reduction in villous branching and trophoblastic proliferation, which ultimately results in nutritional impairment and reduction in oxygen exchange between the mother and fetus.[26]

Similarly, placental thickness was significantly reduced (<2 cm) among the PE and EC cases, which was consistent with findings of other studies.[11,27]

Decreased placental thickness parallels the reduction in the placental weight, which in turn indicates a contemporary failure of villous tissue in a hypoxic environment.

Gross examination revealed placental infarction in more than half of the hypertensive case, consistent with findings of other studies.[16,21] Occlusion of the maternal spiral arterioles and intervillous fibrin deposition leads to localized ischemic necrosis of the villi which ultimately leads to infarction. Studies have emphasized that the extent of infarction directly correlates with disease severity and adverse fetal outcome.[26]

Calcification was observed across all groups; however, it was not statistically significant, aligning with findings of other authors, who suggested that focal calcification represents physiological maturation rather than a pathology. Only diffuse or premature calcification indicates pathological accelerated aging of the placenta.[12]

Retroplacental hematoma, although noted in a subset of diseased and control cases, was not statistically significant which was concordant with findings of other studies, which stated that hematomas may develop secondary to marginal separation unrelated to hypertensive vasculopathy.[12]

Microscopic evaluation of the placenta tissue in our study revealed a significant increase in fibrin deposition, maternal floor infarction and syncytial knotting in PE and EC groups compared with normotensive controls. These findings are consistent with the observation of other studies.[21]

Fibrin deposition

In the present study, score 2 (combined intervillous and perivillous fibrin) was found in 24% of PE and 36% of EC cases, significantly higher than in controls. Excess fibrin deposition reflects maternal vascular malperfusion and trophoblastic hypoxia, leading to a status of maternal blood in the intervillous space. Comparable results were noted by other authors as well.[16,26]

Maternal floor infarction

It was present in multiple foci 34% of PE and 40% of EC cases, compared with 20% of controls. Infarction indicates an ischemic necrosis of villi, which is secondary to obliteration of spiral arteries. The findings were consistent with the findings of other studies.[25,11]

Syncytial knots

They were significantly more frequent in the case groups (84% in OE and 80% in EC) compared with controls. These knots represent an accelerated villous maturation and trophoblastic hyperplasia in response to chronic hypoxia. Similar findings were documented by other authors. [12]

Calcification

Diffuse calcification showed no significant difference between groups, in line with other studies. Physiologic maturation toward term often accounts for focal calcification; only premature and extensive deposits signify pathological aging of the placenta.[28]

Villous basement membrane thickening

Although not statistically significant in this series, it was more common in EC (56%) than PE (44%). Thickening denotes chronic hypoxic fibrosis and collagen deposition, which restricts diffusion across the material fetal interface. Similar trends were described by other authors as well, confirming its association with prolonged maternal hypertension.[7,23]

Objective histopathological scoring

A statistically significant stepwise rise in composite histopathological score was observed from normal (median=4) to PE (median=6) and EC (median=8), demonstrating that structural placental alterations correlate directly with clinical severity. Donthi et al validated a similar semi-quantitative scoring system and proposed it as a reproducible model for standard placental reporting.[21] Gupta et al likewise found that higher histological scores strongly predicted adverse fetal outcomes, emphasizing the diagnostic and prognostic utility of structured morphological assessment.[23]

Collectively, our results reinforce that placental ischemia-related lesions (fibrin, infarction, syncytial knots) serve as morphological correlates of maternal uteroplacental insufficiency. The use of an objective, semiquantitative scoring system enhances inter-observer reproducibility and may aid future clinicopathological correlation and prognostication in hypertensive pregnancies.

Limitations

The present study was conducted at a single tertiary care center with a relatively small sample size, which may limit the generalizability of the findings. Immunohistochemical and molecular markers were not included in the analysis, which could have provided additional insights into the pathophysiological mechanisms. Furthermore, long-term neonatal follow-up was not performed. Future multicentric studies with larger sample sizes and advanced biomarker correlation are recommended to validate the proposed histopathological scoring system.

CONCLUSION

Placental histopathological scoring provides an objective and reproducible means to assess morphological damage in PE and EC. The proposed semi-quantitative scoring system demonstrates a strong correlation between the severity of clinical disease, placental pathology, and

adverse fetal outcomes. This approach may serve as a useful adjunct in evaluating uteroplacental insufficiency and could aid in standardizing placental reporting in HDP. The study contributes to existing knowledge by providing a practical, quantifiable model for correlating placental morphology with clinical outcomes.

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