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PLACENTAL HISTOPATHOLOGICAL CHANGES IN PREECLAMPSIA AND THEIR CORRELATION WITH MATERNAL HEMODYNAMIC RESPONSE TO SPINAL ANAESTHESIA DURING CESAREAN DELIVERY

Dr. Aishwarya Lakshmi Sinha[1], Dr. Harvinder Singh[2], Dr. Saurabh Gupta[3]

[1] Assistant Professor, Department of Obstetrics & Gynaecology, Ananta Charitable Educational Society, Udaipur Rajasthan, India.
[2] Assistant Professor, Department Of Pathology, Prathima Institute Of Medical Sciences, Karimnagar, Telangana, India.
[3] Assistant Professor Department Of Anaesthesia from Rama Medical College, Hospital and Research Centre, Uttar Pradesh, India.

Corresponding Author: Dr. Aishwarya Lakshmi Sinha

Address: Department of Obstetrics & Gynaecology, Ananta Charitable Educational Society, Udaipur Rajasthan, India.

ABSTRACT

Background: Preeclampsia is a multisystem hypertensive disorder of pregnancy associated with abnormal placentation, endothelial dysfunction, and significant maternal and fetal morbidity. Placental histopathological alterations are well recognized in preeclampsia; however, their relationship with maternal hemodynamic response to spinal anaesthesia remains insufficiently explored.

Objectives: To evaluate placental histopathological changes in women with preeclampsia and to determine their correlation with maternal hemodynamic response following spinal anaesthesia during cesarean delivery.

Methods: A hospital-based observational analytical cross-sectional study was conducted at a tertiary care hospital in North India from February 2020 to July 2020. Sixty-six preeclamptic women undergoing cesarean delivery under spinal anaesthesia were enrolled using consecutive sampling. Maternal demographic and obstetric characteristics were recorded. Intraoperative hemodynamic parameters including systolic blood pressure, mean arterial pressure, heart rate, incidence of hypotension, and vasopressor requirement were documented. Placental specimens were subjected to standardized histopathological examination. Associations between placental lesions and maternal hemodynamic outcomes were analyzed using appropriate statistical tests, with $p<0.05$ considered statistically significant.

Results: The mean age of participants was 27.8 ±4.5 years, and 57.6% were primigravidae. Histopathological examination revealed syncytial knot formation in 68.2% of placentas, intervillous fibrin deposition in 59.1%, placental infarction in 51.5%, calcification in 47.0%, accelerated villous maturation in 43.9%, maternal vascular malperfusion in 39.4%, and fibrinoid necrosis in 31.8%. Intraoperative hypotension occurred in 27.3% of patients, while 24.2% required vasopressor support. Placental infarction ($p=0.011$), maternal vascular malperfusion ($p=0.008$), and fibrinoid necrosis ($p=0.015$) were significantly associated with hypotension following spinal anaesthesia. Histopathological severity score demonstrated significant positive correlations with percentage fall in systolic blood pressure ($r=0.41$, $p=0.001$), percentage fall in mean arterial pressure ($r=0.38$, $p=0.003$), and vasopressor requirement ($r=0.35$, $p=0.005$).

Conclusion: Placental vascular and ischemic lesions are frequently encountered in preeclamptic pregnancies and are associated with greater maternal hemodynamic instability following spinal anaesthesia. The severity of placental pathology correlates with the magnitude of blood pressure reduction and vasopressor requirement, suggesting a potential relationship between placental disease burden and maternal cardiovascular response. These findings warrant further investigation in larger prospective studies.

Keywords: Preeclampsia; Placental histopathology; Spinal anaesthesia; Hemodynamic response; Cesarean section; Maternal vascular malperfusion; Placental infarction.

INTRODUCTION

Preeclampsia is a multisystem disorder of pregnancy characterized by new-onset hypertension and proteinuria or evidence of maternal organ dysfunction after 20 weeks of gestation. It remains one of the leading causes of maternal and perinatal morbidity and mortality worldwide, particularly in low- and middle-income countries. The disorder affects approximately 2–8% of pregnancies globally and contributes substantially to adverse maternal outcomes, including eclampsia, cerebrovascular accidents, renal impairment, hepatic dysfunction, and coagulation abnormalities, as well as fetal growth restriction, prematurity, and perinatal death [1,2].

The pathophysiology of preeclampsia is complex and incompletely understood. Current evidence suggests that abnormal placentation during early pregnancy leads to inadequate remodeling of the uterine spiral arteries, resulting in reduced uteroplacental perfusion and chronic placental ischemia [3]. In response to hypoxic stress, the placenta releases antiangiogenic factors, inflammatory mediators, and oxidative stress markers into the maternal circulation, contributing to widespread endothelial dysfunction and altered vascular reactivity [4]. These systemic changes are responsible for the characteristic hemodynamic disturbances observed in preeclamptic women, including increased systemic vascular resistance, altered intravascular volume status, and impaired cardiovascular adaptation to pregnancy [5].

Histopathological examination of the placenta provides valuable insights into the underlying disease process. Several placental lesions have been associated with preeclampsia, including maternal vascular malperfusion, infarction, syncytial knot formation, fibrinoid necrosis, accelerated villous maturation, distal villous hypoplasia, and increased intervillous fibrin deposition [6]. These changes reflect chronic uteroplacental insufficiency and are considered important markers of disease severity and fetal compromise. The degree of placental pathology may also correlate with the extent of maternal endothelial dysfunction and vascular instability [7].

Cesarean delivery is frequently performed in women with severe preeclampsia because of maternal or fetal indications. Spinal anaesthesia is commonly preferred for cesarean section owing to its rapid onset, reliable sensory block, and reduced risks associated with general anaesthesia [8]. However, maternal hemodynamic responses following spinal anaesthesia can be unpredictable in preeclamptic patients. While healthy parturients often experience significant hypotension due to sympathetic blockade, women with preeclampsia may demonstrate a different response because of their elevated baseline vascular tone and altered autonomic regulation [9]. Nevertheless, substantial hemodynamic fluctuations may still occur, potentially affecting maternal and fetal outcomes.

In India, hypertensive disorders of pregnancy continue to constitute a major public health concern and are among the leading contributors to maternal mortality and severe maternal morbidity. Tertiary care hospitals in North India frequently manage high-risk pregnancies complicated by preeclampsia,

yet limited studies have explored the relationship between placental histopathological alterations and maternal cardiovascular responses during spinal anaesthesia in this population [10].

Most available research has focused either on placental pathology or anesthetic outcomes independently, with relatively little attention paid to the potential association between the two. Understanding whether specific placental histopathological changes correlate with maternal hemodynamic responses to spinal anaesthesia may provide additional insights into disease severity, perioperative risk stratification, and individualized anesthetic management. Such information could help clinicians anticipate intraoperative hemodynamic instability and optimize maternal and fetal care.

Therefore, the present study was undertaken to evaluate placental histopathological changes in women with preeclampsia and to assess their correlation with maternal hemodynamic response following spinal anaesthesia during cesarean delivery in a tertiary care hospital in North India. The specific objectives were to describe the spectrum of placental histopathological findings, assess maternal intraoperative hemodynamic parameters after spinal anaesthesia, and determine the association between placental pathological changes and maternal hemodynamic responses.

METHODOLOGY

Study Design: This was a hospital-based observational analytical cross-sectional study conducted to evaluate placental histopathological changes in preeclamptic women and to assess their correlation with maternal hemodynamic response following spinal anaesthesia during cesarean delivery.

Study Setting: The study was conducted in the Department of Anaesthesiology in collaboration with the Departments of Obstetrics & Gynaecology and Pathology at a tertiary care teaching hospital in North India.

Study Duration: The study was conducted from February 2020 to July 2020.

Study Population: The study population comprised pregnant women diagnosed with preeclampsia who underwent cesarean section under spinal anaesthesia during the study period and whose placentas were available for histopathological examination.

Inclusion Criteria

Pregnant women aged 18–40 years.
Singleton pregnancy.
Gestational age ≥28 weeks.
Diagnosis of preeclampsia based on standard obstetric criteria (blood pressure ≥140/90 mmHg measured on two occasions at least 4 hours apart after 20 weeks of gestation with proteinuria and/or evidence of maternal organ dysfunction).
Undergoing elective or emergency lower segment cesarean section under spinal anaesthesia.
Provision of written informed consent.

Exclusion Criteria

Chronic hypertension diagnosed before pregnancy or before 20 weeks of gestation.
Pre-existing cardiovascular disease.
Renal disease unrelated to preeclampsia.
Diabetes mellitus complicating pregnancy.
Multiple gestation.

Placental abnormalities such as placenta previa or placental abruption.
Patients requiring conversion to general anaesthesia.
Inadequate placental tissue for histopathological assessment.
Refusal to participate in the study.

Sample Size: The sample size was calculated using the formula for estimation of a proportion in observational studies:

$$n = \frac{Z^2 \times p \times q}{d^2}$$

Where:

n = required sample size
Z = 1.96 at 95% confidence level
p = anticipated prevalence of significant placental histopathological lesions among preeclamptic pregnancies (assumed 80% based on available literature)
q = 1 − p = 20%
d = absolute precision of 10%

$n = \frac{(1.96)^2 \times 0.80 \times 0.20}{(0.10)^2}]$

n = 61.4

After accounting for possible specimen inadequacy and incomplete data, the final sample size was rounded to 66 participants.

Sampling Technique: A consecutive sampling technique was employed. All eligible preeclamptic women fulfilling the inclusion criteria during the study period were recruited until the desired sample size of 66 was achieved.

Data Collection Tools and Procedure: After obtaining informed written consent, demographic and obstetric information including age, parity, gestational age, booking status, blood pressure measurements, laboratory investigations, and severity of preeclampsia were recorded using a predesigned case record form. Standard monitoring comprising non-invasive blood pressure, electrocardiography, and pulse oximetry was instituted before administration of spinal anaesthesia.

Spinal anaesthesia was administered in the sitting position at the L3–L4 or L4–L5 intervertebral space using hyperbaric bupivacaine according to institutional protocol. Baseline heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure, and oxygen saturation were documented. Hemodynamic parameters were subsequently recorded at predefined intervals intraoperatively. Episodes of hypotension, defined as a reduction in systolic blood pressure of more than 20% from baseline or systolic blood pressure below 90 mmHg, and vasopressor requirements were documented.

Immediately after delivery, placentas were collected and fixed in 10% buffered formalin. Gross examination included placental weight, dimensions, cord insertion, and membrane characteristics. Representative tissue sections were processed and stained with hematoxylin and eosin. Histopathological examination was performed by pathologists blinded to intraoperative hemodynamic findings. Placental lesions including infarction, syncytial knot formation, fibrinoid necrosis, intervillous fibrin deposition, calcification, accelerated villous maturation, and maternal vascular malperfusion were assessed using standard histopathological criteria.

Study Variables: The independent variables included maternal age, parity, gestational age, severity of preeclampsia, placental weight, and histopathological findings such as placental infarction, syncytial knots, fibrinoid necrosis, calcification, intervillous fibrin deposition, accelerated villous maturation, and maternal vascular malperfusion. The dependent variables included maternal hemodynamic response following spinal anaesthesia, specifically changes in systolic blood pressure, diastolic blood pressure, mean arterial pressure, heart rate, incidence of hypotension, and vasopressor requirement.

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using Statistical Package for the Social Sciences (SPSS) software version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were summarized as mean ±standard deviation or median with interquartile range depending on data distribution. Categorical variables were expressed as frequencies and percentages. Comparisons between groups were performed using Student's t-test or Mann–Whitney U test for continuous variables and Chi-square test or Fisher's exact test for categorical variables. Correlations between placental histopathological findings and maternal hemodynamic parameters were assessed using Pearson's or Spearman's correlation coefficients as appropriate. A p-value <0.05 was considered statistically significant.

Ethical Considerations: Written informed consent was obtained from all participants after explaining the objectives, procedures, potential benefits, and confidentiality safeguards. Participation was voluntary, and participants were free to withdraw from the study at any stage without affecting their medical care. Patient confidentiality and anonymity were maintained throughout data collection, analysis, and reporting. The study adhered to the ethical principles outlined in the Declaration of Helsinki and its subsequent amendments.

RESULTS

A total of 66 preeclamptic women undergoing cesarean delivery under spinal anaesthesia were included in the study. The mean maternal age was 27.8 ±4.5 years, and primigravidae constituted more than half of the study population. Severe preeclampsia was present in 43.9% of participants (Table 1).

Table 1. Baseline Maternal and Obstetric Characteristics of the Study Participants (N = 66)

Variable	Value
Age (years), mean ± SD	27.8 ± 4.5
Age ≤25 years, n (%)	24 (36.4)
Age 26–30 years, n (%)	28 (42.4)
Age >30 years, n (%)	14 (21.2)
Primigravida, n (%)	38 (57.6)
Multigravida, n (%)	28 (42.4)
Gestational age at delivery (weeks), mean ± SD	36.1 ± 2.1
Severe preeclampsia, n (%)	29 (43.9)
Non-severe preeclampsia, n (%)	37 (56.1)
Emergency cesarean section, n (%)	41 (62.1)
Elective cesarean section, n (%)	25 (37.9)

Histopathological examination demonstrated a high frequency of placental abnormalities. Syncytial knot formation was the most common lesion, followed by intervillous fibrin deposition, placental

infarction, and calcification. Maternal vascular malperfusion and fibrinoid necrosis were also frequently observed (Table 2).

Table 2. Histopathological Findings in Placental Specimens (N = 66)

Histopathological lesion	Frequency (n)	Percentage (%)
Syncytial knot formation	45	68.2
Intervillous fibrin deposition	39	59.1
Placental infarction	34	51.5
Calcification	31	47.0
Accelerated villous maturation	29	43.9
Maternal vascular malperfusion	26	39.4
Fibrinoid necrosis	21	31.8

Following spinal anaesthesia, a moderate decline in blood pressure was noted. Intraoperative hypotension occurred in 27.3% of patients, while approximately one-fourth required vasopressor support. Bradycardia requiring intervention was uncommon (Table 3).

Table 3. Maternal Hemodynamic Response Following Spinal Anaesthesia (N = 66)

Parameter	Value
Baseline systolic blood pressure (mmHg), mean ± SD	158 ± 14
Lowest intraoperative systolic blood pressure (mmHg), mean ± SD	130 ± 15
Mean reduction in systolic blood pressure (%)	17.5 ± 7.2
Baseline mean arterial pressure (mmHg), mean ± SD	116 ± 11
Lowest mean arterial pressure (mmHg), mean ± SD	96 ± 10
Hypotension after spinal anaesthesia, n (%)	18 (27.3)
Vasopressor requirement, n (%)	16 (24.2)
Bradycardia requiring treatment, n (%)	5 (7.6)

Significant associations were observed between specific placental lesions and intraoperative hypotension. Placental infarction, maternal vascular malperfusion, and fibrinoid necrosis were more common among women who developed hypotension after spinal anaesthesia compared with those who remained hemodynamically stable (Table 4).

Table 4. Association Between Major Placental Lesions and Intraoperative Hypotension

Placental lesion	Hypotension Present (n=18)	Hypotension Absent (n=48)	p-value
Placental infarction	14 (77.8%)	20 (41.7%)	0.011
Maternal vascular malperfusion	12 (66.7%)	14 (29.2%)	0.008
Fibrinoid necrosis	10 (55.6%)	11 (22.9%)	0.015
Syncytial knot formation	15 (83.3%)	30 (62.5%)	0.115

Furthermore, increasing placental histopathological severity was positively correlated with greater reductions in systolic blood pressure and mean arterial pressure, as well as increased vasopressor requirement. No significant correlation was observed between placental histopathological severity and heart rate changes (Table 5).

Table 5. Correlation of Placental Histopathological Severity Score with Maternal Hemodynamic Parameters

Parameter	Correlation Coefficient (r)	p-value
Percentage fall in systolic blood pressure	0.41	0.001
Percentage fall in mean arterial pressure	0.38	0.003
Vasopressor requirement	0.35	0.005
Heart rate change	0.14	0.261

DISCUSSION

The present study evaluated placental histopathological changes in preeclamptic women undergoing cesarean delivery under spinal anaesthesia and examined their association with maternal hemodynamic response following neuraxial blockade. The principal findings were that placental vascular and ischemic lesions were highly prevalent in preeclamptic pregnancies, intraoperative hypotension occurred in approximately one-quarter of patients, and specific placental abnormalities—particularly placental infarction, maternal vascular malperfusion, and fibrinoid necrosis—were significantly associated with greater hemodynamic instability following spinal anaesthesia. Additionally, increasing severity of placental pathological changes correlated with larger reductions in systolic blood pressure and mean arterial pressure as well as increased vasopressor requirement.

Preeclampsia is fundamentally a disorder of abnormal placentation and maternal endothelial dysfunction. The placental lesions observed in the present study are consistent with the established pathological spectrum of maternal vascular malperfusion described in preeclamptic pregnancies [3,6,7]. Syncytial knot formation was the most frequently identified lesion, followed by intervillous fibrin deposition and placental infarction. These findings are in agreement with previous histopathological studies that reported increased syncytial knots, infarcts, fibrinoid necrosis, and accelerated villous maturation in placentas from women with preeclampsia compared with normotensive pregnancies [6,7]. Such lesions are considered morphological manifestations of chronic uteroplacental hypoperfusion resulting from inadequate trophoblastic invasion and impaired spiral artery remodeling [3].

The prevalence of placental infarction in the current study was comparable to observations reported in earlier investigations of hypertensive disorders of pregnancy. Placental infarction reflects significant maternal vascular compromise and reduced perfusion of the intervillous space. Similarly, maternal vascular malperfusion and fibrinoid necrosis are recognized indicators of severe placental ischemic injury and have been associated with adverse maternal and neonatal outcomes [6,7]. The coexistence of these lesions in a substantial proportion of our study population supports the concept that preeclampsia represents a continuum of placental vascular pathology rather than a single pathological process.

The incidence of hypotension following spinal anaesthesia observed in this study was lower than that typically reported among healthy parturients undergoing cesarean section. This finding is consistent with previous evidence suggesting that women with preeclampsia experience less profound

hypotension after spinal anaesthesia because of their increased baseline systemic vascular resistance and heightened vasoconstrictor state [9]. A systematic review and meta-analysis by Aya et al. demonstrated that preeclamptic women are less likely to develop severe spinal anaesthesia-induced hypotension compared with normotensive pregnant women [9]. Nevertheless, a clinically important proportion of patients in our study still experienced hypotension and required vasopressor support, emphasizing the need for careful intraoperative monitoring.

One of the most notable findings of the present study was the significant association between placental vascular lesions and maternal hemodynamic instability. Women exhibiting placental infarction, maternal vascular malperfusion, and fibrinoid necrosis showed a greater likelihood of developing hypotension after spinal anaesthesia. Although direct evidence linking placental histopathology with anesthetic hemodynamic response remains limited, the observed relationship is biologically plausible. Severe placental vascular pathology reflects more advanced endothelial dysfunction and altered cardiovascular adaptation, which may influence vascular responsiveness during sympathetic blockade induced by spinal anaesthesia [4,5].

The positive correlation between histopathological severity and magnitude of blood pressure reduction further supports this hypothesis. Placental pathology may therefore serve as a surrogate marker of the underlying systemic disease burden in preeclampsia. While placental examination is inherently retrospective, recognition of these associations may improve understanding of disease mechanisms and contribute to future risk stratification models.[10]

From a clinical perspective, the findings highlight the importance of integrated multidisciplinary management of preeclamptic patients undergoing cesarean delivery. Awareness that severe placental vascular pathology may reflect greater susceptibility to intraoperative hemodynamic fluctuations could assist anesthesiologists in optimizing perioperative monitoring, fluid management, and vasopressor preparedness. Furthermore, the study reinforces the value of placental histopathological evaluation as an important component of quality obstetric care and clinical audit in hypertensive pregnancies.

The strengths of this study include the prospective collection of perioperative hemodynamic data, standardized histopathological assessment of placental specimens, and evaluation of an underexplored relationship between placental pathology and anesthetic outcomes. The study also involved collaboration among obstetrics, anaesthesiology, and pathology departments, enhancing the comprehensiveness of the analysis.

However, several limitations should be acknowledged. First, the study was conducted at a single tertiary care center, which may limit generalizability. Second, the sample size was relatively modest and may have reduced the ability to detect weaker associations. Third, the absence of a normotensive control group precluded direct comparison of placental pathology and hemodynamic responses between hypertensive and non-hypertensive pregnancies. Finally, placental histopathological assessment, although standardized, remains subject to some degree of observer variability. Larger multicenter studies are required to validate these findings and further clarify the mechanisms linking placental pathology and maternal cardiovascular responses during spinal anaesthesia.

CONCLUSION

This study demonstrated that placental histopathological abnormalities are common in preeclamptic pregnancies undergoing cesarean delivery under spinal anaesthesia. The most frequently observed lesions were syncytial knot formation, intervillous fibrin deposition, placental infarction, and calcification, reflecting underlying uteroplacental vascular insufficiency. Maternal hemodynamic changes following spinal anaesthesia were generally well tolerated; however, a significant proportion of patients developed intraoperative hypotension and required vasopressor support. Importantly, placental infarction, maternal vascular malperfusion, and fibrinoid necrosis were significantly associated with hemodynamic instability after spinal anaesthesia. Furthermore, increasing severity of placental histopathological changes correlated with greater reductions in blood pressure and higher vasopressor requirements. These findings suggest that placental vascular pathology may reflect the extent of systemic disease involvement in preeclampsia and may be associated with maternal cardiovascular responses during neuraxial anaesthesia.

Further multicenter studies with larger sample sizes are recommended to validate these observations and explore their potential role in perioperative risk assessment and individualized anesthetic management of women with preeclampsia.

DECLARATIONS

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Conflict of Interest: The authors declare that there is no conflict of interest.

Informed Consent: Written informed consent was obtained from all participants prior to enrollment in the study.

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