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Original Research Article

A Morphological Study of Placenta and Umbilical Cord in Pre-Eclampsia Females

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Abstract:

Background: One of the main causes of maternal and perinatal morbidity, especially in developing countries like India, is pre-eclampsia, a multisystem hypertensive disease of pregnancy. The uteroplacental ischemia that characterizes this syndrome causes the placenta, which serves as the vital physiological interface between the mother and the fetus, to undergo major structural changes.

Objective: This study's main goal was to assess and correlate the gross morphometric characteristics and histological alterations of the placenta and umbilical cord in women who had been diagnosed with pre-eclampsia at a tertiary care facility in Bihar's Seemanchal region.

Methods: In this study, 106 placentas from pre-eclamptic women were examined at Katihar Medical College and Hospital's Department of anatomy and Obstetrics & Gynecology. Placental weight, diameter, thickness, and umbilical cord insertion were among the gross parameters that were carefully measured. A thorough histological inspection of tissue slices stained with hematoxylin and eosin to pinpoint certain ischemia lesions came next.

Results: In comparison to normative criteria, the study found a statistically significant decrease in mean placental weight (412.5 ± 58.2 g). Gross abnormalities were common, with retroplacental hematomas occurring in 14.1% of cases and placental infarction occurring in 48.1% of cases. The prevalence of non-central insertions in the umbilical cord was high (60.2%). Histologically, there was a significant rise in fibrinoid necrosis (45.2%), cytotrophoblastic proliferation, and syncytial knots (62.2%), all of which are signs of hypoxia and accelerated placental aging.

Conclusion: Pre-eclampsia is linked to severe placental maldevelopment, as demonstrated by the morphological changes found in this study. The results of significant ischemia damage and decreased placental mass highlight the importance of placental evaluation in comprehending the pathophysiology of unfavorable fetal outcomes in hypertension pregnancies.

Introduction

Pre-eclampsia is a complicated syndrome unique to pregnancy that remains a significant obstetric problem worldwide. Clinically, it is characterized by the emergence of hypertension (blood pressure ≥140/90 mmHg) and proteinuria in a previously normotensive woman after the twentieth week of pregnancy [1]. Pre-eclampsia continues to be a major cause of maternal death and iatrogenic preterm delivery despite advancements in prenatal care, especially in countries with limited resources. The incidence is thought to account for 8–10% of all pregnancies in India, which greatly increases the burden of newborn illness [2]. The etiology is strongly established in the placenta, even if the clinical presentation involves several maternal organ systems, such as the brain, kidneys, and liver. The placenta's crucial involvement in the disorder's pathophysiology is shown by the fact that it is still the only effective treatment.

The placenta is commonly referred to as the "diary of intrauterine existence" or the "mirror of the fetus." It is a highly adaptable organ that changes its form to suit the maternal environment in order to ensure fetal life. In pregnancies exacerbated by hypertension, the placenta takes the brunt of ischemia stress, resulting in a variety of morphological and histological abnormalities. Retrospective evaluation of these changes provides a unique window into the pregnancy's pathophysiology, which can help explain unfavorable outcomes such as intrauterine growth restriction (IUGR) and stillbirths. While the Western literature contains a wealth of information on

placental pathology, there is a notable absence of detailed morphological research from Bihar's seemanchal region, which is distinguished by distinctive dietary, socioeconomic, and genetic characteristics.

Pathophysiology of Placental Insufficiency: The basic flaw in pre-eclampsia is largely recognized to be the failure of the second wave of trophoblastic invasion. In a normal pregnancy, extravillous cytotrophoblasts enter the maternal spiral arteries and replace the muscular and elastic tunica medium with fibrinoid tissue. This physiological remodeling changes the spiral arteries from high-resistance, low-flow channels to low-resistance, high-flow conduits capable of supporting the developing embryo. According to Roberts and Hubel's "two-stage concept," pre-eclampsia starts with reduced placental perfusion (Stage 1) caused by shallow trophoblastic invasion [3]. This causes placental ischemia, oxidative stress, and the release of anti-angiogenic factors (such sFlt-1) into the maternal blood. These variables generate widespread maternal endothelial dysfunction (stage 2), which leads to the clinical syndrome of hypertension and proteinuria.

Clinical Significance of Morphological Assessment: Understanding the shape of the placenta is not only of academic interest but also has significant clinical implications. Gross parameters like placental weight and diameter are direct predictors of the functional placental volume available for food exchange; a tiny placenta always results in a small infant. Furthermore, specific gross lesions, such as retroplacental hematomas or severe infarction, might cause abrupt fetal discomfort or intrauterine death. Histopathological characteristics such as enlarged syncytial knots (Tenney-Parker alterations) and basement membrane thickening act as tissue biomarkers for the duration and severity of the ischemia. By combining these morphological findings with clinical data, clinicians might gain a better understanding of the recurrence risk in future pregnancies, as well as the long-term cardiovascular health consequences for both mother and child.

Aim and Objectives: The major goal of this study was to do a detailed morphological assessment of placentas from pre-eclamptic women who delivered at Katihar Medical College and Hospital. Specific aims were:

1. To measure gross morphometric indices such as weight, dimension, and thickness.
2. To identify and measure macroscopic ischemia lesions, including infarcts and retroplacental clots.
3. To categorize umbilical cord anomalies based on insertion places.
4. To document and connect microscopic structural changes in chorionic villi with the pathophysiology of placental ischemia.

Materials and Methods

Study Design and Setting: The current investigation was designed as a retrospective observational study in a hospital setting. It was carried out collaboratively by the Departments of anatomy and Obstetrics and Gynecology at Katihar Medical College and Hospital in Bihar. This tertiary care center serves a large rural and semi-urban population, resulting in a diversified sample set that is representative of the area demographic profile. The 18-month research covered cases handled between January 2023 and June 2024. Before data collection began, the Institutional Ethics Committee evaluated and approved the study protocol.

Sample Selection and Ethical Considerations: The final analysis comprised a total of 106 placentas. The screening approach was stringent to verify that the findings were only due to pre-eclampsia rather than any confounding comorbidities.

- **Inclusion Criteria:** Women who satisfied the diagnostic criteria for pre-eclampsia, defined as a blood pressure measurement of ≥140/90 mmHg on two occasions at least 4 hours apart, accompanied with proteinuria (≥1+ on dipstick) appearing after the 20th week of gestation, were included in the study.
- **Exclusion Criteria:** We firmly omitted cases involving multiple pregnancies (twins/triplets), because placentation in these circumstances differed dramatically from that in singletons. Women with a history of chronic hypertension, pre-existing or prenatal diabetes mellitus, severe anemia (Hemoglobin < 7 g/dL), active TORCH infections, or clinical indications of chorioamnionitis were excluded to ensure sample homogeneity.

Gross Processing and Morphometry: Placental specimens were processed according to the standardized techniques provided by Benirschke and Kaufmann [4]. Following delivery, the placentas were placed in clean, labeled containers and transferred to the histopathology lab. If quick examination was not possible, specimens were preserved in 10% neutral buffered formalin. The first procedure entailed carefully washing the placenta under running tap water to remove surface blood and clots, ensuring that the real parenchymal weight was recorded.

The umbilical chord was clamped and cut at a standard distance of 5 cm from the insertion point. The membranes were carefully separated from the placental border. Weighing was done with a high-

precision electronic balance. For dimensional analysis, the greatest diameter and diameter perpendicular to it were measured with a metallic ruler, and the mean of these two values was recorded as the placental diameter. Thickness was measured by putting a long, graded needle into the center of the placenta and a peripheral site, then taking the average of these depths.

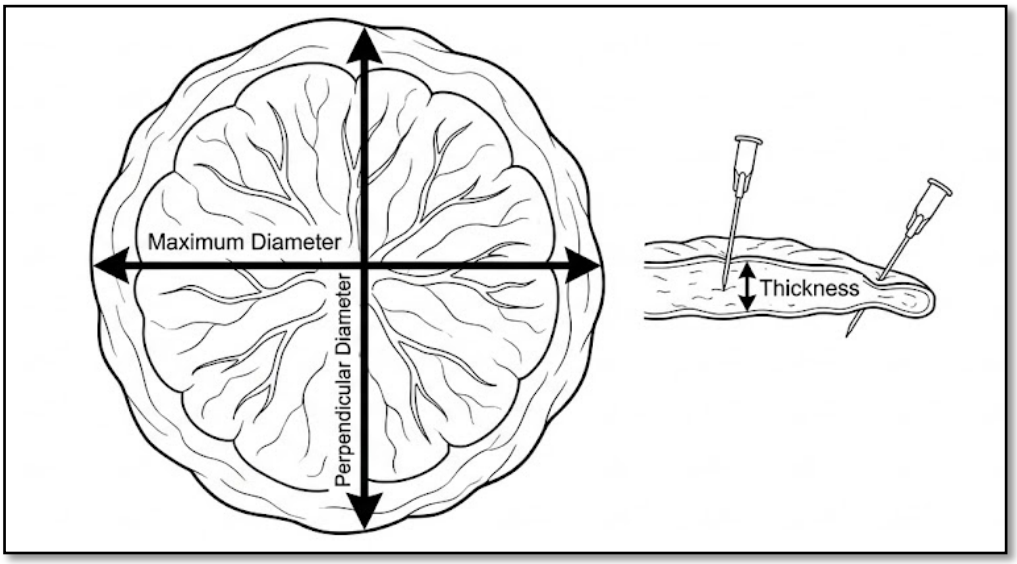

Figure 1: **Gross Placental Measurement Techniques**

Macroscopic and Histopathological Evaluation: The maternal surface was carefully examined for the number of cotyledons and the presence of localized lesions. We specifically searched for areas of infarction, which appear as pale, firm, white patches, as well as retroplacental clots that indicate an abruption. Calcification was diagnosed by a gritty sensation when palpated and sectioned.

Tissue blocks were carefully selected for histopathological examination. Full-thickness sections were taken from the center zone, the peripheral perimeter, and any locations with obvious pathologies such as infarctions or hematomas. These tissue blocks were subjected to standard processing, which included dehydration in graded alcohols, xylene clearing, and paraffin wax embedding. A rotary microtome was used to cut sections to a thickness of 4-5 microns, which were then stained with hematoxylin and eosin. Two independent pathologists examined the slides using a light microscope to assess villous vascularity, the presence of syncytial knots, fibrinoid necrosis, and villous basement membrane thickening.

Results

Maternal Demographics: According to the research population, the illness is most common among younger women. The participants had an average age of 24.6 ± 3.8 years. Primigravidae made up the majority of the cohort, accounting for roughly 68% of the cases, which is consistent with the widely held belief that the immunological risk of pre-eclampsia is highest during the first pregnancy. The average gestational age at delivery was 36.4 ± 2.1 weeks, highlighting the need for preterm induction in hypertensive pregnancies to avoid maternal problems.

Gross Morphological Findings: The morphometric study of the placentae revealed a widespread restriction in placental growth. The average placental weight was 412.5 ± 58.2 g, with a range of 290 g to 540 g. This average weight is significantly less than the typical 500 g expected for a healthy term placenta. The average diameter was 15.8 ± 1.9 cm, with a mean thickness of 1.9 ± 0.4 cm.

This cohort had a high incidence of macroscopic lesions. Placental infarction was the most prevalent anomaly, affecting nearly half of the patients (48.1%). The locations of these infarcts varied, but they were mostly in the margins. Retroplacental bleeding, shown as adhering clots or depressed areas on the maternal surface, was discovered in 14.1% of the placentas. Gross calcification was perceptible in 29.2% of the specimens.

Table 1: **Gross Morphometric Parameters of Placenta (n=106)**

Parameter	Mean ± SD	Range
Placental Weight (g)	412.5 ± 58.2	290 – 540
Diameter (cm)	15.8 ± 1.9	12 – 19
Thickness (cm)	1.9 ± 0.4	1.4 – 2.8
Number of Cotyledons	16.2 ± 3.1	10 – 22

Umbilical Cord Morphology: The average length of the umbilical chord was 48.2 ± 10.5 cm. The high proportion of aberrant insertion locations was a surprising observation. While central insertion is considered the typical, it was found in just 39.6% of the instances (42 women). The bulk of cords had non-central insertion points. Eccentric insertion was the most prevalent, accounting for 42.4% of cases. Marginal insertion, also known as the Battledore placenta, was found in 16.9% of the individuals.

Table 2: **Distribution of Umbilical Cord Insertion Sites**

Type of Insertion	Number of Cases (n=106)	Percentage (%)
Central	42	39.6%
Eccentric	45	42.4%
Marginal (Battledore)	18	16.9%
Velamentous	1	0.9%

Histopathological Assessment: Microscopic inspection of the H&E-stained sections revealed significant structural alteration of the chorionic villi. The most common feature was the presence of more syncytial knots, which were seen in 62.2% of cases. Fibrinoid necrosis was another notable observation, occurring in 45.2% of the samples and frequently interrupting the continuity of the villous structure. Furthermore, 36.8% of the placentas showed cytotrophoblast growth as a compensatory mechanism.

Table 3: **Histopathological Changes in Placental Villi**

Histological Feature	Number of Cases (n=106)	Percentage (%)
Increased Syncytial Knots	66	62.2%
Fibrinoid Necrosis	48	45.2%
Cytotrophoblastic Proliferation	39	36.8%
Villous Basement Membrane Thickening	34	32.0%
Atherosis of Spiral Arteries	12	11.3%
Calcification	31	29.2%

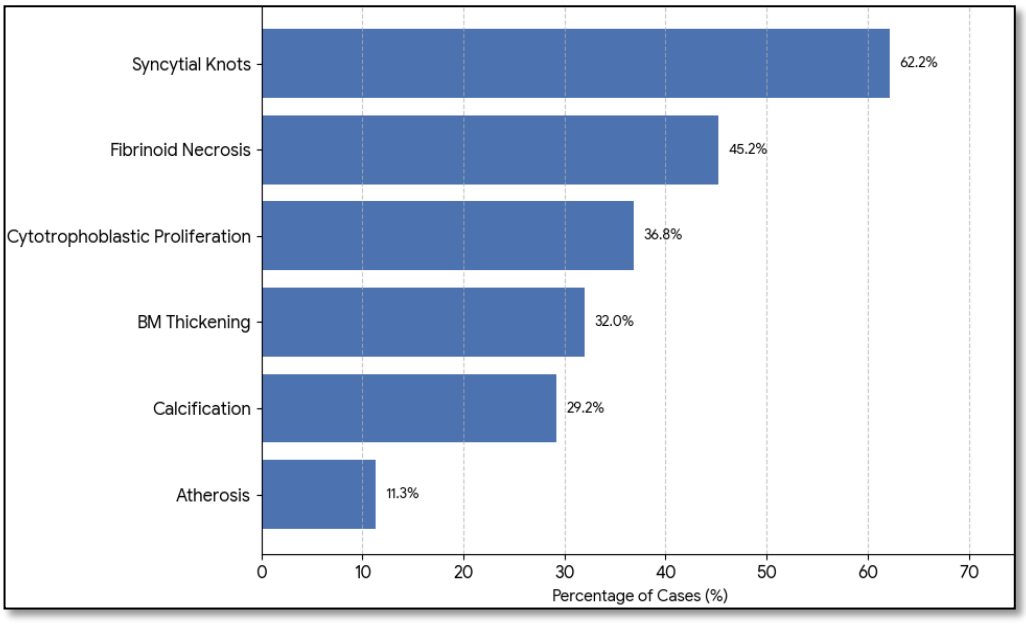

Figure 2: **Frequency of various histopathological changes observed**

Discussion

The placenta acts as a functional biological archive, recording stress and adaptations to the intrauterine environment throughout gestation. In the case of pre-eclampsia, the organ experiences chronic hypoperfusion, resulting in structural changes that are visible both grossly and microscopically. This study of 106 placentas from Katihar Medical College provides important regional data supporting the relationship between hypertension diseases and placental abnormalities.

Morphometric Analysis and Placental Efficiency: Our data on placental weight are consistent with the idea of placental growth restriction. Our observed mean weight of 412.5 g is much lower than the conventional reference for term pregnancies and is consistent with the findings of Udainia et al., who reported similar decreases in eclamptic individuals [5]. The explanation for this weight loss is most likely twofold: first, the failure of spiral artery remodeling limits the availability of nutrients needed for placental tissue growth; second, severe infarction and ischemia necrosis result in a loss of functional parenchymal mass. This decrease in placental mass is an important predictor of fetal weight because a smaller placenta has less surface area for the exchange of oxygen and nutrients. The lower diameter and thickness support the concept of a "starved" placenta that does not realize its full morphological potential.

Interpretation of Macroscopic Lesions: The significant prevalence of placental infarction (48.1%) in our study can be attributed to the vascular pathology associated with pre-eclampsia. While tiny, marginal infarctions can occur in normal pregnancies, the infarctions seen in pre-eclampsia are frequently larger and more central. According to Fox et al., infarctions encompassing more than 5% of the placental tissue are pathogenic and closely related with fetal hypoxia [6]. These lesions indicate regions where the blood supply has been totally cut off, rendering that portion of the placenta useless.

In a similar vein, it is concerning that retroplacental hematomas occur in 14.1% of cases. The severe disorder known as placental abruption, in which the placenta prematurely separates from the uterine wall, is typified by this lesion. These hematomas are more likely to occur when the decidual vessels burst due to the vascular damage in pre-eclampsia. Our results highlight the ongoing risk of abruption in this patient group and are in line with those of Khan et al., who found a similar incidence rate in hypertensive pregnancies [7].

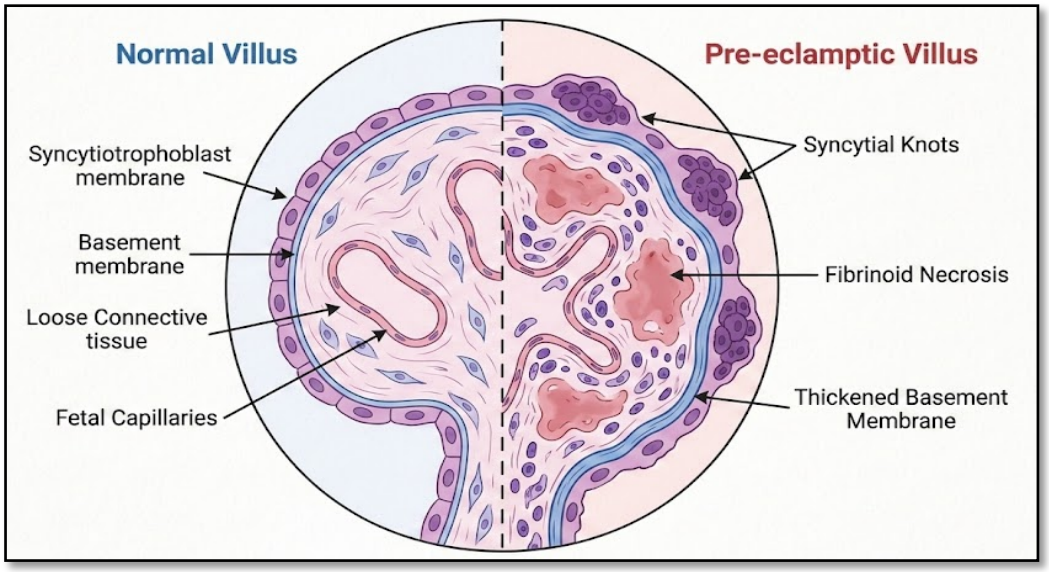

Figure 3 Histopathological Changes in Pre-Eclampsia

Umbilical Cord Anomalies and Trophotropism: The study found a large variation in umbilical cord insertion sites, with more than 60% of instances exhibiting eccentric or marginal insertion. This is significantly greater than the general population rate. The notion of "trophotropism" proposed by Rath et al. provides a persuasive explanation for this occurrence [8]. It implies that the placenta actively migrates or develops preferentially to locations with superior blood supply. In pre-eclampsia, when the uterine environment is hostile and inadequately perfused, the placenta may grow asymmetrically to reach more vascularized decidua, leaving the original cord insertion point at the periphery. Marginal insertions are clinically risky because the vessels are less covered by Wharton's jelly, rendering them more vulnerable to compression during delivery, which can cause acute fetal discomfort.

Histopathological Correlations: In pre-eclampsia, the placenta shows signs of "accelerated maturation." The presence of enlarged syncytial knots in 62.2% of our instances is the defining feature of this process, also known as Tenney-Parker alterations. While knots are typical of term placentas, their abundance in pre-eclampsia reflects a reaction to hypoxia [9]. In locations with limited perfusion, the syncytiotrophoblast nuclei combine to lower metabolic demand.

Furthermore, the proliferation of cytotrophoblasts (Langhans cells) seen in 36.8% of instances suggests a regenerative attempt. Under normal circumstances, cytotrophoblasts diminish as pregnancy progresses. Their return or persistence in pre-eclampsia indicates an attempt to heal the damaged syncytial layer [10]. However, parallel thickening of the basement membrane undermines this repair by increasing the diffusion distance for oxygen, exacerbating fetal hypoxia. The presence of fibrinoid necrosis indicates immune-mediated damage and turbulent flow inside the intervillous region.

Comparison with Existing Literature: Our findings are fairly consistent with those of other research conducted in the Indian subcontinent. For instance, similar placental weight reductions were noted by Majumdar et al. in Kolkata, who attributed them to the disease process and the mother's nutritional status [11]. The incidence of calcification in our sample (29.2%) is comparable to the 30% reported by Ahmed and Daver [12], suggesting that the disease is consistently associated with premature placental aging across all demographic groups. Additionally, our results about the correlation between the degree of hypertension and morphological alterations align with Redline's comprehensive analyses [13], which emphasize that maternal vascular malperfusion is a particular pathological feature of pre-eclampsia.

Conclusion

The current retrospective study, undertaken at Katihar Medical College and Hospital, provides solid evidence of the negative consequences of pre-eclampsia on placental shape. The statistics show that pre-eclampsia is more than just a maternal disease; it is profoundly entrenched in placental maldevelopment. We found a statistically significant reduction in placental gross dimensions and weight, which is a crude but efficient indicator of placental insufficiency. The high prevalence of ischemic lesions, including as infarctions and retroplacental hematomas, is directly proportional to the severity of vascular maladaptation.

Furthermore, the study provides insight into the microscopic environment of the pre-eclamptic placenta. The high frequency of syncytial knots, fibrinoid necrosis, and basement membrane thickening indicates that the organ is experiencing various levels of oxidative stress and accelerated aging. These structural changes limit the fetomaternal exchange surface, which provides a pathological explanation for the intrauterine growth restriction and fetal distress that are typical in these pregnancies.

Gross and microscopic examination of the placenta remains a vital tool in rural and semi-urban India, where modern molecular diagnostics may not always be available. It permits obstetricians and pathologists to reconstruct the intrauterine events that resulted in negative outcomes. We urge that routine histological evaluation of the placenta be made mandatory in all cases of hypertensive pregnancy problems. This approach will not only help with immediate newborn management, but it will also provide important data for counseling women about the hazards of future pregnancies. Future prospective studies with bigger sample sizes and long-term neonatal follow-up are required to better understand the therapeutic significance of these morphological changes.

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