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EVALUATION OF PLACENTAL PATHOLOGIES IN HYPERTENSIVE VS. NORMOTENSIVE PREGNANCIES: A CROSS-SECTIONAL STUDY

Original Research

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ABSTRACT

Background: Hypertensive disorders of pregnancy are a leading cause of maternal and fetal morbidity and mortality worldwide. These conditions are closely linked to placental abnormalities that may compromise fetal development and contribute to adverse pregnancy outcomes.

Objective: To compare the occurrence and types of placental abnormalities in hypertensive versus normotensive pregnant women at the time of delivery.

Methods: This cross-sectional study was conducted over eight months in a tertiary care hospital in Lahore, Pakistan. A total of 200 pregnant women were enrolled, including 100 with hypertensive disorders and 100 normotensive controls. Placentas were collected immediately after delivery for gross and histopathological examination. Parameters such as placental weight, infarctions, hematomas, calcifications, and microscopic lesions including syncytial knots, villous infarctions, and vascular malperfusion were evaluated. Data were analyzed using SPSS version 26. Independent t-tests and chi-square tests were used for statistical comparisons, with p-values <0.05 considered significant.

Results: Hypertensive pregnancies exhibited significantly more placental abnormalities compared to normotensive ones. Mean placental weight was lower in the hypertensive group (437.5g vs. 496.2g). Rates of infarctions (42% vs. 15%), hematomas (19% vs. 6%), and calcifications (51% vs. 28%) were notably higher. Histologically, increased syncytial knots (67% vs. 23%), villous infarctions (54% vs. 16%), and maternal vascular malperfusion (63% vs. 19%) were significantly more prevalent in hypertensive pregnancies.

Conclusion: Hypertensive disorders are associated with significant structural and vascular changes in the placenta, which may underlie poor perinatal outcomes. Placental evaluation in such pregnancies is essential for understanding disease impact and guiding clinical management.

Keywords: Calcification, Hypertension, Infarction, Placenta, Pregnancy Complications, Preeclampsia, Syncytial Knots.

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INTRODUCTION

Hypertensive disorders in pregnancy remain among the leading causes of maternal and perinatal morbidity and mortality worldwide. Affecting up to 10% of pregnancies globally, these disorders include conditions such as gestational hypertension, chronic hypertension, preeclampsia, and eclampsia. The adverse effects of maternal hypertension are not limited to the mother alone; they extend to the fetus, often mediated through alterations in placental structure and function (1). As the critical interface between the mother and fetus, the placenta plays a central role in ensuring fetal growth and development, making it a valuable focus of investigation in understanding the pathophysiology of hypertensive pregnancies. The placenta is a complex organ responsible for nutrient transfer, waste elimination, gas exchange, and endocrine functions essential to a successful pregnancy. Any compromise in its development or function can have significant consequences for both maternal and fetal outcomes (2). In hypertensive pregnancies, abnormal placentation is frequently observed, potentially driven by inadequate trophoblastic invasion and defective spiral artery remodeling. These changes may lead to reduced uteroplacental perfusion and are often associated with a spectrum of pathological findings in the placenta, including infarctions, retroplacental hematomas, increased syncytial knots, villous hypoplasia, and abnormal vasculature (3).

Several studies have explored the relationship between maternal hypertension and placental pathology. For instance, research has shown that preeclampsia is associated with a higher prevalence of placental infarcts, decidual arteriopathy, and increased fibrin deposition. In contrast, normotensive pregnancies are generally associated with fewer such abnormalities, suggesting that the hypertensive milieu uniquely influences placental morphology (4,5). Nevertheless, while this link is recognized, much of the existing literature has focused on specific forms of hypertensive disorders or has lacked a direct comparative analysis between hypertensive and normotensive pregnancies within a unified study framework (6). This lack of comprehensive, comparative research represents a critical gap in the understanding of how maternal blood pressure status impacts placental pathology at term. Without robust, side-by-side evaluations of hypertensive versus normotensive pregnancies, clinicians and researchers remain limited in their ability to interpret placental findings meaningfully in the context of maternal blood pressure status. Furthermore, such data are essential for informing risk stratification, antenatal surveillance strategies, and delivery planning, particularly in settings with limited access to advanced diagnostic tools (7,8).

Given this background, a systematic and comparative analysis of placental pathology in hypertensive and normotensive pregnancies is warranted. Cross-sectional studies examining placental tissues at the time of delivery offer a valuable opportunity to evaluate the cumulative impact of maternal hypertension on placental health (9,10). By identifying specific histopathological patterns associated with hypertensive states, such research can improve understanding of disease mechanisms, support early diagnosis, and potentially guide interventions aimed at improving pregnancy outcomes. In this context, the current study seeks to address this important clinical and scientific need by comparing the occurrence and types of placental abnormalities in women with hypertensive versus normotensive pregnancies. The objective is to evaluate whether hypertensive disorders are significantly associated with higher rates or distinct types of placental pathology at delivery, thereby contributing to a more nuanced understanding of how maternal blood pressure influences placental health and fetal well-being.

METHODS

This cross-sectional study was conducted over a period of eight months at the Department of Obstetrics and Gynecology in a tertiary care teaching hospital in Lahore, Pakistan, known for serving a diverse obstetric population across both urban and semi-urban communities. The primary objective was to compare the frequency and types of placental abnormalities observed in hypertensive versus normotensive pregnant women at the time of delivery. The study design was chosen to allow for a simultaneous assessment of maternal blood pressure status and placental pathology at delivery, ensuring a snapshot of outcomes across a defined population. Based on prevalence rates from previous literature, with an assumed frequency of placental abnormalities of 50% in hypertensive pregnancies and 30% in normotensive pregnancies, a minimum sample size was calculated using the OpenEpi tool. At 95% confidence level and 80% power, with a 1:1 ratio between groups, the minimum sample size required was 174 participants—87 in each group. To account for non-respondents or poor-quality placental specimens, the sample was increased to 200 women, comprising 100 hypertensive and 100 normotensive participants selected consecutively through non-probability purposive sampling (11).

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Participants were recruited at the time of delivery in the labor ward and operation theatre. Inclusion criteria for the hypertensive group included pregnant women aged 18–40 years with a singleton pregnancy, who were diagnosed with any form of hypertensive disorder in pregnancy (including gestational hypertension, chronic hypertension, preeclampsia, or eclampsia) according to the American College of Obstetricians and Gynecologists (ACOG) guidelines. Normotensive participants were matched for gestational age and parity and had consistently normal blood pressure readings throughout pregnancy. Exclusion criteria for both groups included women with diabetes mellitus, known renal disease, autoimmune disorders, or fetal anomalies, as these conditions may independently influence placental morphology (11,12). Upon delivery, placentas were collected fresh and immediately transported to the pathology department in a sterile container. Gross examination was conducted within two hours of delivery, assessing parameters such as placental weight, shape, cord insertion site, and presence of infarctions, hematomas, or calcifications. Each placenta was then fixed in 10% buffered formalin for 24 hours before histopathological processing. Standard hematoxylin and eosin staining techniques were applied, and microscopic evaluation was conducted by two independent histopathologists blinded to maternal clinical status to reduce observer bias.

Histopathological variables examined included syncytial knots, villous infarction, fibrinoid necrosis, villitis, intervillous thrombi, chorangiosis, and maternal vascular malperfusion lesions. These variables were selected based on prior literature indicating their relevance in hypertensive disorders of pregnancy. A structured proforma was used to document each finding, ensuring consistency and completeness of data capture. Data were compiled and analyzed using IBM SPSS version 26. Descriptive statistics were employed to summarize baseline demographic and obstetric characteristics. Mean and standard deviation were calculated for continuous variables, while categorical variables were expressed as frequencies and percentages. The Shapiro-Wilk test confirmed normal distribution of continuous data. Independent sample t-tests were used to compare continuous variables between groups, such as placental weight and size. Categorical data, including histopathological findings, were analyzed using the chi-square test or Fisher's exact test, where appropriate. A p-value of less than 0.05 was considered statistically significant.

Ethical approval was obtained from the Institutional Review Board (IRB) of the hospital prior to the initiation of the study. Written informed consent was obtained from all participants after a thorough explanation of the study's purpose, procedures, and confidentiality safeguards. Participation was entirely voluntary, and participants were assured that their clinical care would not be affected by their decision to participate or withdraw from the study. All research procedures conformed to the principles outlined in the Declaration of Helsinki. Rigorous attention was paid to maintaining the integrity and anonymity of all patient data throughout the study duration. The methodological framework of this study was carefully structured to ensure scientific validity, ethical rigor, and replicability, providing a meaningful contribution to the understanding of how hypertensive conditions in pregnancy affect placental pathology at term.

RESULTS

A total of 200 placentas were examined, including 100 from hypertensive and 100 from normotensive pregnancies. The mean age of participants in the hypertensive group was slightly higher compared to the normotensive group. The gestational age at delivery was also lower among hypertensive women, with a greater proportion undergoing cesarean sections. Baseline demographic data are detailed in Table 1. Gross placental examination revealed notable differences between the groups. The mean placental weight was significantly lower in the hypertensive group (437.5 g) compared to the normotensive group (496.2 g). Visible infarctions were present in 42% of hypertensive placentas versus 15% in normotensive ones. Retroplacental hematomas were observed in 19% of hypertensive cases, notably higher than the 6% found in normotensive pregnancies. Calcifications occurred more frequently in the hypertensive group as well (51% vs. 28%). These findings are presented in Table 2 and illustrated in Figure 1. Histopathological assessment revealed that increased syncytial knots were identified in 67% of hypertensive placentas, in contrast to 23% in normotensive samples. Villous infarctions were seen in more than half of the hypertensive group (54%) compared to just 16% among normotensive controls. Fibrinoid necrosis and maternal vascular malperfusion lesions were also markedly more common in hypertensive pregnancies (47% vs. 12% and 63% vs. 19%, respectively). These trends are documented in Table 3 and visually represented in Figure 2. Microscopic lesions such as chorangiosis, intervillous thrombi, and villitis were observed more frequently in the hypertensive group. Specifically, chorangiosis was detected in 29% of hypertensive placentas, compared to 11% in normotensive ones. Intervillous thrombi occurred in 21% versus 9%, and villitis in 18% versus 10%, respectively. These results are summarized in Table 4. These findings collectively indicate a higher prevalence and severity of gross and microscopic placental abnormalities in hypertensive pregnancies compared to normotensive ones.

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Table 1: Demographics of Study Participants

Variable	Hypertensive Group (n=100)	Normotensive Group (n=100)
Mean Age (years)	29.4	27.8
Gestational Age at Delivery (weeks)	36.1	38.3
Primigravida (%)	45 (45%)	52 (52%)
Multiparous (%)	55 (55%)	48 (48%)
Mean BMI (kg/m²)	28.2	26.7
Cesarean Delivery (%)	68 (68%)	44 (44%)

Table 2: Gross Placental Features

Gross Feature	Hypertensive Group (n=100)	Normotensive Group (n=100)
Mean Placental Weight (g)	437.5	496.2
Visible Infarctions (%)	42 (42%)	15 (15%)
Retroplacental Hematoma (%)	19 (19%)	6 (6%)
Calcifications (%)	51 (51%)	28 (28%)

Table 3: Histopathological Findings

Histopathological Feature	Hypertensive Group (n=100)	Normotensive Group (n=100)
Increased Syncytial Knots (%)	67 (67%)	23 (23%)
Villous Infarctions (%)	54 (54%)	16 (16%)
Fibrinoid Necrosis (%)	47 (47%)	12 (12%)
Maternal Vascular Malperfusion (%)	63 (63%)	19 (19%)

Table 4: Other Microscopic Lesions

Lesion	Hypertensive Group (n=100)	Normotensive Group (n=100)
Chorangiosis (%)	29 (29%)	11 (11%)
Intervillous Thrombi (%)	21 (21%)	9 (9%)
Villitis (%)	18 (18%)	10 (10%)

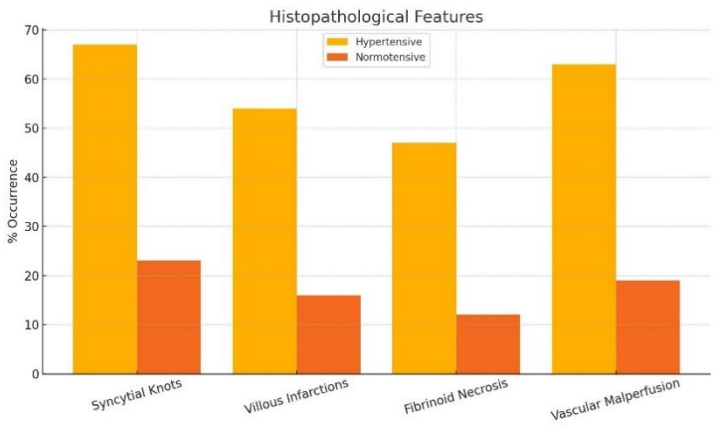


Figure 1 Histopathological Features

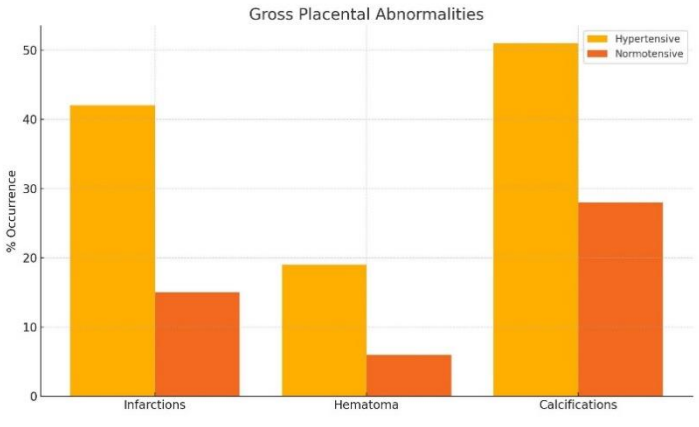


Figure 2 Gross Placental Abnormalities

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DISCUSSION

The present study demonstrated a significantly higher occurrence of both gross and histopathological placental abnormalities in hypertensive pregnancies compared to normotensive ones. These findings align with a growing body of literature suggesting that hypertensive disorders of pregnancy lead to impaired placental development and function, contributing to adverse maternal and fetal outcomes. Gross morphological changes, such as reduced placental weight and increased incidences of infarctions, hematomas, and calcifications, were markedly more prevalent in hypertensive pregnancies. Similar patterns were reported, significant decreases in placental weight, volume, and diameter among hypertensive mothers, along with a greater frequency of infarctions and calcifications (13,14). Likewise, a study found that hypertensive placentas showed notable reductions in size and surface area and a greater number of cotyledon abnormalities and thrombotic lesions (15). Histologically, the hypertensive group exhibited higher rates of syncytial knot formation, villous infarctions, fibrinoid necrosis, and maternal vascular malperfusion. These findings are strongly supported by multiple studies. Studies reported similar histopathological alterations, attributing them to hypoxic damage resulting from inadequate spiral artery remodeling and reduced uteroplacental blood flow (16,17). These structural changes compromise nutrient and oxygen exchange, directly contributing to fetal growth restriction and preterm delivery.

The presence of secondary microscopic lesions, such as chorangiosis and intervillous thrombi, also supports the hypothesis of placental hypoxia and chronic underperfusion. These findings resonate with those of a study which found a significant increase in syncytial knots, fibrinoid necrosis, and stromal fibrosis in hypertensive pregnancies (18,19). Additionally, a study introduced novel insights into the protective role of extracellular vesicles in normotensive pregnancies, suggesting that their absence or dysregulation may explain the pronounced vascular lesions seen in hypertensive placentas (20).

Despite the compelling findings, this study has certain limitations. Being cross-sectional in design, it can establish associations but not causation. The sample size, although calculated for statistical power, may not capture the full spectrum of placental pathology across diverse hypertensive subtypes. Additionally, histopathological interpretations, although performed by blinded experts, may involve some degree of subjectivity. The exclusion of confounding comorbidities, while necessary for homogeneity, may limit the generalizability of the results to all pregnant populations. Nevertheless, the study holds several strengths. The prospective design and consistent methodology for placental examination enhance the reliability of findings. The use of both gross and microscopic assessments offers a comprehensive view of the structural disruptions associated with hypertensive disorders. Furthermore, conducting the study in a tertiary care setting improves its applicability to real-world clinical environments where high-risk pregnancies are managed.

In light of these findings, the clinical implications are clear. Routine placental examination in hypertensive pregnancies could serve as a valuable tool for postnatal assessment and may offer retrospective insight into fetal growth restriction or adverse perinatal events. Furthermore, antenatal prediction of such abnormalities using non-invasive imaging modalities remains an area of ongoing research. Recent studies have explored ultrasonographic placental grading and location as potential early indicators of preeclampsia, emphasizing their potential in antenatal risk stratification (21,22). Future research should aim to explore molecular and functional biomarkers in placental tissue that may predict maternal and fetal outcomes. Longitudinal studies with larger and more diverse populations would also help in validating and refining the observed associations. Integrating placental pathology with clinical parameters and maternal-fetal biomarkers could enhance risk prediction and guide individualized obstetric care.

CONCLUSION

This study demonstrated a significantly higher frequency of gross and histopathological placental abnormalities in hypertensive pregnancies compared to normotensive ones. The findings highlight the impact of maternal hypertension on placental structure, which may contribute to adverse perinatal outcomes. Routine placental examination in such cases may offer valuable clinical insights and support tailored antenatal care to improve maternal-fetal health.

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AUTHOR CONTRIBUTION

Author	Contribution
Shua Nasir*	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published
Shazia Sultana	Substantial Contribution to study design, acquisition and interpretation of Data Critical Review and Manuscript Writing Has given Final Approval of the version to be published
Kainat Zulfiqar	Substantial Contribution to acquisition and interpretation of Data Has given Final Approval of the version to be published
Kiran Rafique	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Syeda Fatima Rizvi	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Shahzada Khalid Sohail	Substantial Contribution to study design and Data Analysis Has given Final Approval of the version to be published
Sangeen Khan Tareen	Substantial Contribution to study design and Data Analysis Has given Final Approval of the version to be published

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