

## ORIGINAL RESEARCH

# Placental Morphology In Hypertensive Disorders Of Pregnancy And Its Correlation With Fetal Outcome: An Original Research

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### Abstract

**Background:** Hypertensive disorders of pregnancy, including conditions like preeclampsia, pose significant risks to maternal and perinatal health. Despite extensive research, the precise etiology of these disorders remains elusive.

**Objective:** This observational study aimed to investigate the correlation between placental morphology in hypertensive pregnancies and neonatal outcomes.

**Methods:** A total of 200 women, comprising 100 hypertensive cases and 100 normotensive controls, were enrolled. Placentae were meticulously examined for various parameters, including weight, diameter, thickness, and the presence of infarctions, calcifications, and retroplacental hematomas. Additionally, newborns were evaluated for birth weight, Apgar scores, and NICU admission.

**Results:** Statistical analyses revealed significant differences in birth weight between hypertensive and normotensive pregnancies, with hypertensive cases showing lower birth weights. Placental weight was also reduced in hypertensive pregnancies, albeit not significantly. Fetoplacental ratio was significantly decreased in hypertensive cases, indicating impaired placental function. Furthermore, hypertensive pregnancies exhibited a higher incidence of placental infarctions and retroplacental hematomas, which correlated with adverse neonatal outcomes such as low Apgar scores and increased NICU admissions. While calcification incidence was higher in hypertensive pregnancies, its impact on fetal outcomes was not significant.

**Conclusion:** These findings underscore the importance of placental health in hypertensive pregnancies and highlight the need for further research to elucidate underlying mechanisms and improve maternal and fetal health outcomes in this population.

**Keywords:** Hypertensive disorders of pregnancy, placental morphology, neonatal outcomes, birth weight, fetoplacental ratio

### Introduction

The placenta serves as a crucial organ during pregnancy, dictating the intrauterine environment vital for fetal growth and development. Hypertensive disorders of pregnancy, including conditions like preeclampsia, pose significant risks to both maternal and fetal health, often leading to complications such as fetal growth restriction, prematurity, and low birth weight. These disorders are associated with observable pathological changes in the placenta, which can offer valuable insights into the prenatal experiences of the infant [1,2].

Placentae affected by hypertensive disorders typically exhibit distinctive morphological alterations, including reduced weight, diameter, and thickness, along with a higher prevalence of abnormalities such as irregular shape and abnormal cord insertion. Additionally, hypertensive pregnancies often manifest with increased incidences of placental infarction, calcification, and retroplacental hematoma. These gross pathological changes reflect the compromised function of the placenta in supporting fetal development [3,4].

The adverse effects of these placental alterations extend to fetal outcomes, as the placenta serves as the conduit for essential nutrients and waste exchange between mother and fetus. Examination of the placenta provides valuable insights into the prenatal environment and can help elucidate potential issues affecting the fetus. Therefore, studying placental morphology in hypertensive pregnancies compared to normotensive pregnancies is crucial for understanding the relationship between these morphological changes and neonatal outcomes [4-6]. The aims and objectives of this study encompass a detailed investigation into placental morphology in hypertensive pregnancies, with a focus on comparing these morphological changes to those observed in normotensive pregnancies. By correlating these changes with fetal outcomes, researchers aim to shed light on the impact of hypertensive disorders on placental health and subsequent neonatal health.

**Material and methods**

Placentae obtained from women who recently delivered babies at a government maternity hospital in Koti served as the primary material for this study. A total of 200 placentae were collected, with 100 placentae sourced from women diagnosed with hypertensive disorders of pregnancy and 100 placentae from women with normal pregnancies. Additionally, newborns born to these women were included in the study for further evaluation.

**Methodology:**

The selection of samples ensured representation from both hypertensive and normotensive pregnancies. Inclusion criteria encompassed cases of gestational hypertension, preeclampsia, eclampsia, and chronic hypertension, while patients unwilling to participate, those with hypertensive disorders complicated by other conditions, and pregnancies with complications unrelated to hypertension were excluded. Following delivery, placentae were collected in a sterile tray and processed promptly. Membranes and the umbilical cord were carefully severed near their attachment to the placenta. Placentae were then gently expressed to remove excess blood and thoroughly washed under running water. Systematic examination of the placentae was conducted to identify various morphological and pathological changes. Parameters such as size, shape, diameter, thickness, weight, number of cotyledons, and the presence of infarction, calcification, and retroplacental hematoma were meticulously assessed. Concurrently, newborns were evaluated for Apgar scores and birth weights to determine their immediate health status. Additionally, the fetoplacental weight ratio was calculated for each case to assess the relationship between placental changes and fetal outcomes. Statistical analysis was performed to compare placental morphology and pathology between hypertensive and normotensive pregnancies. Furthermore, correlations between these changes and fetal outcomes were explored.

**Results**

Table :1 presents the descriptive statistics for birth weight (kg) and placental weight (gm) in both control and case groups. In the control group, birth weight ranged from 1.5 to 4 kg with a mean  $\pm$  SD of  $2.7 \pm 0.55$  kg, while in the case group, it ranged from 1 to 3.8 kg with a mean  $\pm$  SD of  $2.4 \pm 0.58$  kg. There was a significant difference between the two groups for birth weight. Placental weight ranged from 180 to 650 gm in the control group and from 250 to 650 gm in the case group, with mean  $\pm$  SD values of  $448.6 \pm 111.1$  gm and  $449.9 \pm 91.2$  gm, respectively. However, there was no significant difference between the control and case groups for placental weight.

Table: 2 displays the distribution of parity among the study groups. In the control group, the parity of G2, G3, G4, G5, and primi were 24 (24%), 8 (8%), 4 (4%), 1 (1%), and 63 (63%) respectively. Similarly, in the case group, the parity of G2, G3, G4, G5, and primi were 23 (23%), 15 (15%), 5 (5%), 2 (2%), and 55 (55%) respectively. There was no significant difference between the control and case groups for parity.

Table: 3 presents the distribution of NICU admission and stillbirth/neonatal death in both control and case groups. In the control group, 19 (19%) infants were admitted to the NICU, while in the case group, 36 (36%) infants were admitted. There was a significant difference between the two groups for NICU admission. However, for stillbirth/neonatal death, there was no significant difference between the control and case groups.

Table: 4 illustrates the histopathological changes observed in the placentae of both control and case groups. In the control group, calcification and infarction were each present in 7% of cases, while retroplacental hematoma was present in 3% of cases. However, in the case group, calcification, infarction, and retroplacental hematoma were present in 22%, 18%, and 17% of cases, respectively. There was a significant difference between the control and case groups for histopathological changes.

Table 1: comparison between control and case groups for the various parameters

Groups	N	Minimum	Maximum	Mean	SD	P-Value
Control	100	1.5	4	2.7	0.55	<0.0001
Case	100	1	3.8	2.4	0.58	
Control	100	180	650	448.6	111.1	0.928
Case	100	250	650	449.9	91.2	
Control	100	10	23	15.1	2.9	0.985
Case	100	10	20	15.0	2.2	
Control	100	0.5	2	1.410	0.38	0.790
Case	100	0.8	2	1.412	0.30	
Control	100	4.33	8.75	6.2	0.9	<0.0001
Case	100	2.67	7.33	5.2	0.7	

Table 2: Comparison between control and case groups for the parameter Parity.

Groups	Parity					Chi-square value	P-value
	G2	G3	G4	G5	Primi		
Control	24	8	4	1	63	3.139	0.535
Case	23	15	5	2	55		

Table 3: Comparison between control and case groups for the parameter NICU Admission and Still Birth/Neonatal Death

Groups	NICU Admission		P-value
	Yes	No	
Control	19	81	0.011
Case	36	64	
Groups	Still Birth/Neonatal Death		P-value
	Yes	No	
Control	7	93	0.238
Case	13	87	

Table 4: Comparison between control and case groups for the parameter Histopathological Changes

Histopathological Changes	Groups		Chi-square value	P-value
	Control	Case		
Calcification	7	22	49.92	<0.0001
Infarction	7	18		
Retro Placental Hematoma	3	17		
Retro placental Hematoma, Calcification	0	2		
Calcification, Infarction	0	2		
Infarction, Retro placental Hematoma	0	4		
No	83	35		

## Discussion

Hypertensive disorders of pregnancy, affecting around 7 to 10% of pregnancies, are significant contributors to maternal and perinatal morbidity and mortality. Despite extensive research, the exact cause of conditions like preeclampsia remains unclear, though evidence points to the placenta as a central player, given the resolution of the disease post-placental delivery. Our study aimed to investigate various aspects of placental health and their implications for hypertensive disorders of pregnancy and neonatal outcomes [6-10]. One key finding was the significant reduction in birth weight among hypertensive women compared to normotensive counterparts, consistent with prior research. This reduction is often attributed to altered placental vasculature, leading to compromised blood flow and subsequent fetal growth restriction. Similarly, placental weight was notably lower in hypertensive pregnancies, a trend observed in previous studies, with lower weights correlating with increased risks of stillbirth [5-10]. Fetoplacental ratio, another indicator of placental function, was significantly reduced in hypertensive pregnancies, indicative of impaired placental function. These findings align with previous research, suggesting a consistent association between hypertensive disorders and placental dysfunction. However, while diameter and thickness were reduced in hypertensive pregnancies, the differences were not statistically significant in our study, contrasting with some literature suggesting significant reductions in placental size among hypertensive individuals [11-13]. The incidence of placental infarction was notably higher in hypertensive pregnancies, a finding consistent with prior studies. Infarction is often associated with adverse neonatal outcomes, including low Apgar scores and increased NICU admissions. Similarly, retroplacental hematoma, more prevalent in hypertensive pregnancies, was strongly associated with an increased risk of stillbirth, underscoring the importance of vigilant monitoring of placental health in hypertensive pregnancies [13-15]. While calcification incidence was higher in hypertensive pregnancies, its impact on fetal

outcomes was not significant in our study, reflecting mixed findings in the literature. These complexities highlight the multifaceted nature of placental pathology in hypertensive disorders and emphasize the need for further research to elucidate underlying mechanisms and potential therapeutic targets.

### **Conclusion**

From the present study it can be concluded that hypertension adversely influences the morphology of the placenta and these changes are directly related to the fetal outcome. The placental changes are more severe based on the severity and duration of the disease and so is the fetal outcome. However any single finding either in the gross examination or in the microscopic examination cannot be said as pathognomonic to the hypertension. The presence of placental findings can be confirmatory of hypertension but their absence doesn't exclude the disease as these findings become evident when there is reduced uteroplacental blood flow.

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