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Correlation of Placental Changes with Fetal and Maternal Outcome in Mothers with Hypertensive Disorders of Pregnancy

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ABSTRACT:

Background:

Hypertension represents one of the most prevalent complications encountered during pregnancy, significantly contributing to both maternal and perinatal morbidity and mortality. The placenta, acting as a crucial feto-maternal organ, plays a pivotal role in sustaining pregnancy and fostering the healthy development of the fetus. Notably, the weight of the placenta holds functional importance, as it correlates with the villous surface area and fetal metabolism. Motivated by this context, the present study endeavors to examine the morphological and histological alterations in the placenta associated with hypertensive disorders of pregnancy and to assess their correlation with maternal and fetal outcomes.

Methods: A prospective investigation encompassing 120 cases was conducted, comprising 60 normotensive mothers (Group A) and 60 mothers with hypertensive disorders of pregnancy (Group B). Placental specimens obtained from these participants were subjected to morphological and histological analyses, with subsequent correlation with maternal and fetal outcomes.

Results: Our findings revealed a notable reduction in placental weight and dimensions within Group B compared to Group A. Histopathological examination unveiled a significant increase in the incidence of syncytial knots, fibrinoid necrosis, hyalinization, and calcification in placentas from the hypertensive group, directly correlating with neonatal complications. Furthermore, the mean neonatal birth weight was significantly higher in Group A compared to Group B.

Conclusion: Hypertensive disorders of pregnancy exert a substantial impact on placental morphology, leading to diminished weight and dimensions, indicative of placental insufficiency attributable to compromised utero-placental blood flow. These alterations ultimately influence neonatal weight and overall neonatal outcomes. Our study underscores the presence of distinct morphological changes in the placenta that detrimentally affect fetal growth.

KEYWORDS: Hypertensive disorder of pregnancy, Fibrinoid necrosis, Syncytial knots, Placental insufficiency, Intrauterine growth restriction

INTRODUCTION

Hypertension stands as a formidable complication in pregnancy, afflicting approximately 5-10% of pregnancies and forming a significant part of the deadly triad of maternal morbidity and mortality alongside hemorrhage and infections. [1] The impact of hypertensive disorders of pregnancy on the placenta is profound, resulting in reduced weight and dimensions, and altering its histology, thereby adversely affecting fetal growth. [2]

Anomalies in placental vasculature development are considered fundamental in the pathophysiology of preeclampsia, leading to adverse fetal outcomes. Defective implantation characterized by incomplete extravillous trophoblastic invasion of the spiral arteriolar wall, a hallmark of preeclampsia, results in small caliber vessels with high resistance flow, thus compromising placental blood flow and oxygenation, affecting fetal growth. Typically, the extent of inadequate trophoblastic invasion is believed to be associated with the intensity of the hypertensive condition. Although abnormal invasion of trophoblast may be the causative event in less than 20% cases it is still considered significant. [3]

Preeclampsia, prematurity and idiopathic intrauterine growth retardation each often show abnormal incomplete or failed uterine vascular adaptation resulting in variable persistence of vascular muscle and elastic lamina. [4, 5]

Syncytial knot formation, indicative of hormonal disturbances, may further contribute to altered placental morphology and the occurrence of hypertensive disorders in mothers and low birth weight in babies. Scarred, shrunken, fibrotic and hypovascular villi with reduced number or caliber of placental capillaries are seen due to destruction of growing villous capillaries by abnormal uteroplacental flow and is evidenced by fetomaternal hemorrhage in second trimester.[6, 7]

Highly toxic radicals that injure endothelial cells, modify their nitric oxide production and interfere with prostaglandin balance. Moreover, it decreases endothelial nitric oxide synthase expression thus increasing nitric oxide inactivation. [8] Other consequences of oxidative stress include production of the lipid-laden macrophage foam cells seen in atherosis. Damaged or activated endothelial cells may produce less nitric oxide and secrete substances that promote coagulation and increase sensitivity to vasopressors. [9]

In hypertensive disorders of pregnancy the terminal villous differentiation is disturbed with truncation of distal villi, alteration of arrangement of intra-cotyledon vasculature, and vasculopathy of the spiral arteries; leading to decreased blood flow and hypoxia.[10] Under hypoxic conditions, redistribution of blood flow to the most critical organs such as the brain and the heart of the IUGR fetus ensues. This blood redistribution further reduces placental flow and increases vascular resistance which interferes with foetal nutrition and growth, leading to a decrease in neonatal weight, foetal distress, and death.

Through this study, we aim to delve into the intricate morphological and histological changes within the placenta of mothers afflicted with hypertensive disorders during pregnancy, unraveling the interconnected effects on both maternal and fetal health.

MATERIAL AND METHODS

A hospital-based, cross-sectional study was conducted from June 2022 to January 2024 at the Department of Obstetrics and Gynaecology in SUT Academy of Medical Sciences, located in Vencode, Vattapara, Thiruvananthapuram, Kerala, India. We approached eligible women attending the ANC clinic or admitted to the labour room for participation, with written informed consent obtained from willing participants. The study included only third-trimester mothers without comorbidities such as diabetes mellitus, GDM, hypothyroidism, or epilepsy. A total of 120 pregnant women were enrolled, divided into 60 with hypertensive disorders of pregnancy (case group) and 60 normotensive mothers (control group). We took a detailed medical history, performed obstetric examination, and conducted placental evaluations.

Placental specimens were collected post-delivery and subjected to gross and microscopic examinations. Maternal outcomes, including delivery mode and complications, and fetal outcomes, such as birth weight and NICU admission, were recorded. Statistical analyses, including Spearman and Pearson correlations, were performed using SPSS 17 software to assess the correlation between placental changes and maternal and fetal outcomes.

RESULT

A significant decrease in placental weight was observed in the case group compared to controls ($P < 0.001$), with weights decreasing progressively from gestational hypertension (426.36g) to preeclampsia (335.53g) and eclampsia (215g). There was a significant decrease in placental diameter in hypertensive mothers ($P < 0.001$), and a strong correlation with placental weight (Pearson correlation = 0.85). The number of cotyledons varied from 14-25 in all groups, with no significant association found between the number of cotyledons and the incidence of hypertensive disorders of pregnancy ($P = 0.212$). Similarly, we observed no significant association between the incidence of hypertensive disorders of pregnancy and the site of umbilical cord insertion. The case group exhibited higher incidences of syncytial knot, fibrinoid necrosis, hyalinization, calcification, and retroplacental clots ($P < 0.001$). We also noticed moderate negative correlations between placental weight and these histological features and a strong correlation with placental diameter, except for retroplacental clots, which showed a weak negative correlation with placental weight and no correlation with placental diameter.

Significant increases in cesarean section and preterm vaginal deliveries were observed in mothers with hypertensive disorders of pregnancy, accompanied by a notable reduction in full-term vaginal deliveries. The mean gestational age at delivery was significantly lower (p value <0.001) in hypertensive mothers (36.63 weeks) compared to normotensive mothers (38.87 weeks), with gestational age decreasing progressively from gestational hypertension (37.91 weeks) to preeclampsia (36.32 weeks) to eclampsia (32.25 weeks). In this study, 5 hypertensive mothers required admission to the MICU out of which 3 had eclampsia and 2 had preeclampsia. The mean neonatal birth weight was significantly reduced in the case study group (2313.33gms) in comparison with normotensive mother (3027.17gms). (P value- 0.000). Twenty-one neonates in the hypertensive group required NICU admission, while none in the control group did, indicating a significant increase in NICU admissions among infants born to hypertensive mothers (P value < 0.001). Additionally, neonates of hypertensive mothers exhibited poorer mean APGAR scores (6.60/8.25) compared to those of normotensive mothers (8.03/9.88) (P value < 0.001). Two mothers with preeclampsia experienced intrauterine fetal demise. There was no significant correlation of placental changes with poor maternal outcome, except for a weak positive correlation with retroplacental clots. However, placental changes showed a moderate correlation with poor neonatal outcomes, except for no correlation with retroplacental clots.

Placental changes to Placental weight	Pearson correlation
Syncitial knot	-0.528
Fibrinoid necrosis	-0.528
Calcification	-0.471
Hyalinisation	-0.528
Retroplacental clots	-0.354

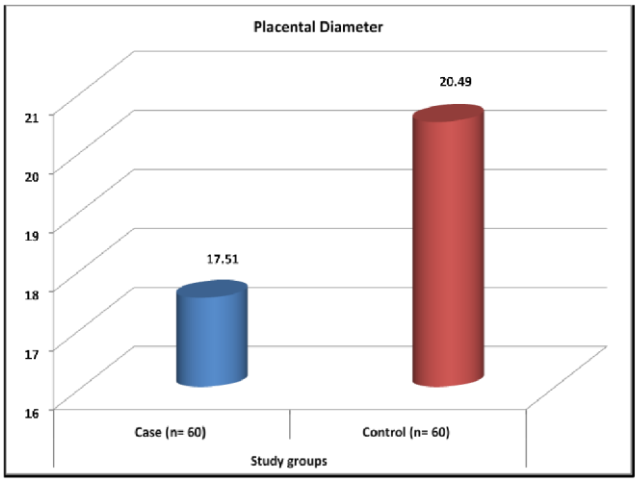

Fig I: Placental diameters in case and control group

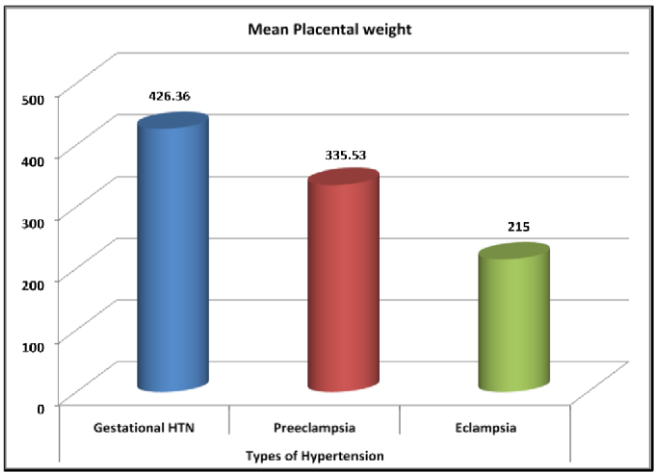

Fig II: Placental weight in increasing severity of hypertension

Placental changes to Placental diameter	Pearson Correlation
Syncitial knot	-0.632
Fibrinoid necrosis	-0.632
Calcification	-0.486
Hyalinisation	-0.632
Retroplacental clots	-0.272

Fig III: Correlation with placental changes and placental weight

Placental changes to Poor Maternal outcome	Spearman Correlation
Syncitial knot	0.200
Fibrinoid necrosis	0.200
Calcification	0.209

Hyalinisation	0.200
Retroplacental clots	0.300

Fig IV: Correlation with placental changes and poor maternal outcome

Placental changes to Poor Neonatal outcome	Spearman Correlation
Syncitial knot	0.405
Fibrinoid necrosis	0.405
Calcification	0.424
Hyalinisation	0.405
Retroplacental clots	0.222

Fig V: Correlation with placental changes and poor neonatal outcome

DISCUSSION

Hypertensive disorders represent the most prevalent medical complication of pregnancy and stand as a leading cause of perinatal and maternal morbidity, second only to embolism in maternal mortality [1]. The classical etiopathogenesis of these disorders implicates placental and utero-placental insufficiency, evident in profound morphological and histological changes within the placenta, reflecting the hazards that the fetus undergoes during intrauterine growth and development.

In our study, the placental weights in the study groups were consistently below 500g, with the lowest recorded weight at 140g, findings consistent with those reported by various authors such as Mallik et al., Nobis and Das, and Bhatia et al., where minimum placental weights ranged from 300-200g [11, 12, 13]. Spiral artery vasculopathy leads to reduced blood flow, consequently resulting in decreased placental weight, as described by Robertson [10]. This reduced maternal uteroplacental insufficiency indirectly causes fetal vessel constriction.

The mean placental diameter in our study was notably smaller in hypertensive mothers compared to normotensive ones, suggesting an underlying pathological cause interfering with normal placental growth, as observed in previous studies by Mallik et al. and Cibils [11, 14]. Additionally, in our study, placental weights decreased progressively with increasing severity of hypertensive disorders of pregnancy.

Histological examination of the placenta revealed various structural changes, such as a significant number of syncytial knots, areas of fibrinoid necrosis, calcification, and hyalinization, all of which showed a strong correlation with placental diameter and weight [15]. Microscopic findings, like localized fibrinoid necrosis and hyalinization, may depict placental mosaicism, potentially contributing to placental insufficiency and fetal growth retardation [16]. Furthermore, there was a significant increase in the number of syncytial knots in the placenta, indicating hormonal disturbance and altered blood flow leading to hypertensive disorders of pregnancy and low birth weight babies. Our present study also demonstrates a moderate correlation between placental changes and neonatal complications, with weak to no correlation observed with maternal complications. In addition, 15 placentas in the case study group had retroplacental clots, primarily associated with eclampsia and preeclampsia.

Regarding cord insertion patterns, our study found no significant correlation between insertion pattern and the incidence of hypertensive disorders of pregnancy. Similarly, the number of cotyledons varied in our study, ranging from 14-25, with no significant correlation observed between the number of cotyledons and the presence of hypertensive disorders of pregnancy.

CONCLUSION

Hypertensive diseases of pregnancy still remains a major problem in developing countries. Our study sheds light on the intricate relationship between hypertensive disorders of pregnancy and placental morphology. The observed reductions in placental weight and diameter, coupled with histological changes such as syncytial knots and fibrinoid necrosis, underscore the pathological impact of these disorders. These findings highlight the importance of placental evaluation in the management of hypertensive pregnancies and emphasize the need for further research to elucidate underlying mechanisms and potential therapeutic targets.

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