Available online on <u>www.ijpcr.com</u>

International Journal of Pharmaceutical and Clinical Research 2024; 16(5); 511-516

Original Research Article

Study on Morphology of Placenta and Fetal Outcomes in Hypertensive Pregnancies

Anjani Kumari¹, Shilpi Singh², Deepshikha Singh³, Seema⁴

¹Senior Resident, Department of Obstetrics and Gynaecology, Darbhanga Medical College and Hospital, Laheriasarai, Bihar

²Senior Resident, Department of Obstetrics and Gynaecology, Darbhanga Medical College and Hospital, Laheriasarai, Bihar

³Tutor, Department of Anatomy, All India Institute of Medical Sciences, Patna, Bihar ⁴Professor and Head of Department, Department of Obstetrics and Gynaecology, Darbhanga Medical College and Hospital, Laheriasarai, Bihar

Received: 25-02-2024 / Revised: 23-03-2024 / Accepted: 26-04-2024 Corresponding Author: Dr. Shilpi Singh Conflict of interest: Nil

Abstract:

Background: The placenta's normal construction, complete development, and functions dictate the survival and growth of the fetus. Study on the placenta provides information on the health of the developing fetus.

Methods: In all, 100 participants were involved in this study, which ran from February 2023 to January 2024 in the Department of Obstetrics and Gynecology at the DMCH in Laheriasarai, Bihar, with assistance from the Department of Anatomy. Participants were divided into two groups: those with hypertension and those without it.

Results: Twenty percent of the females in the hypertensive groups were multigravida, compared to 80% of primigravida females. Preterm births increased significantly (to 35%) and lower section caesarean sections accounted for a sizable part of deliveries (to 45%) in the hypertensive group. The babies' mean birth weights were 2.82 ± 0.43 kg in the control group and 2.33 ± 0.84 kg in the hypertension group. The mean placental weight was found to be $541.4\pm30.62g$ in the control group and $478.4\pm30.62g$ in the hypertension group upon morphometry analysis of the placentas. In one group, the mean placental diameter was 17.87 ± 1.47 cm, whereas in the control group it was 19.87 ± 1.47 cm, comparing the hypertension group to the control group, there was a substantial decrease in placental area, volume, and thickness. Mean number of cotyledons were 18.46 ± 1.54 in control group as against 16.46 ± 1.54 in hypertension group. Fetoplacental ratio and placental coefficient were 5.23 ± 0.86 and 0.19 ± 0.02 in control group and 4.88 ± 1.79 and 0.17 ± 0.06 in the hypertension group.

Conclusion: Pregnancy-related hypertension affects the placenta's shape and morphometry, as well as the health of the developing fetus.

Keywords: Placenta; Fetus; Morphology; Morphometry; Hypertensive Pregnancies.

This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/4.0) and the Budapest Open Access Initiative (http://www.budapestopenaccessinitiative.org/read), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

Introduction

The placenta, a critical organ that connects the mother with the fetus, is one of the two key components of reproduction [1]. The placenta's normal construction, full development, and functions dictate the survival and growth of the fetus [2]. The placenta experiences morphological and morphometric changes during pregnancy, including modifications to its weight, volume, structure, form, and function to support the developing fetus [3].

The placenta reflects the health of the mother and fetus. It is a report on the perinatal experience of the child. Thus, the placenta significantly reflects pregnancy problems that are linked to increased perinatal morbidity and mortality.Preeclampsia is a significant pregnancy complication that affects 5-10% of pregnancies and increases the risk of morbidity and mortality in both the mother and the fetus. It is specific to pregnancy, its genesis is unknown, and it is one of the main causes of death for both mothers and/or fetuses [2, 3].

Proteinurea and hypertension are linked to preeclampsia. Its main cause is the death of cytotrophoblast cells, which results in the superficial penetration of decidua and the escape of 30–50% of spiral arterioles from trophoblast remodeling in the placental bed [3,4]. These arterioles' undilated state is caused by their intact myometrial segments and unaffected adrenergic nerve supply. As a result, there is less opportunity for the freely communicating sinusoids that are necessary for healthy blood flow. The mean luminal diameter of uterine spiral arterioles in preeclamptic women is found to be less than onethird that of normal pregnancies. As gestation progresses, there is a consequent decrease in uteroplacental perfusion and placental infarction, which leads to fetal hypoxia and ultimately IUGR, which is linked to preterm birth and fetal death [5, 6].

A thorough assessment of the placenta provides information that can help control difficulties for both the mother and the infant [6,9].

This study was done keeping these things in mind. The goal of the current study is to evaluate the morphology and histology of placentas from mothers who have prostate cancer and compare the results with those from pregnancies that are typically uneventful.

Material and Methods

From February 2023 to January 2024, the Department of Obstetrics and Gynecology at

Darbhanga Medical College and Hospital in Laheriasarai, Bihar, collaborated with the Department of Anatomy to conduct this prospective, observational study.

This study comprised 100 patients with hypertensive pregnancies (test group) and 100 subjects with normotensive pregnancies (control group), as well as normotensive moms and mothers with gestational hypertension and pregnancyinduced hypertension (preeclampsia and eclampsia).

This study excluded all other maternal diseases that result in limited placental size, placental infarcts, and intrauterine growth retardation.

Results

200 patients, aged 20 to 35, were assessed for placental characteristics in the labor room of the department of obstetrics and gynecology and the anatomy department of the DMCH, Laheriasarai, Bihar. Of these, 100 were in the normotensive group and the remaining 100 were in the hypertension group (Table 1).

Table 1: Distribution of cases

Normotensive		Hyper	tensive group						
group		Gesta	tional Hypertension	on Pre-eclampsia		Eclampsia		Total	
No.	Percentage	No.	Percentage	No.	Percentage	No.	Percentage	No.	Percentage
100	100%	25	25%	50	50%	25	25%	100	100%

The age range of 20–25 years old accounted for the majority of cases (46.2%) in the control group, while the age group of 26–30 years old accounted for the majority of cases (42.4%) in the hypertension group. 12% (n=12) of the normotensive group were multigravida, while 88% (n=88) were primigravida. Conversely, 20% (n=20) of the females in the hypertensive groups were multigravida, and 80% (n=80) of the females were primigravida (Table 2).

Gravida status	Normotensive group		Hypertensive group		
	No.	Percentage	No.	Percentage	
Primigravida	88	88%	80	80%	
Multi gravida	12	12%	20	20%	
Total	100	100%	100		

 Table 2: Gravida status of selected patients

The control group's blood pressure was within normal bounds. The average blood pressure measured was 123.07 ± 4.14 mmHg for the systolic and 82.31 ± 4.08 mmHg for the diastolic phases. Most patients in the hypertensive group had elevated blood pressure on both the systolic and diastolic levels, whereas some only had elevated systolic and few just elevated diastolic readings.

In the hypertensive group, the mean diastolic blood pressure was 91.14 ± 4.14 mmHg, and the mean systolic blood pressure was 149.8 ± 7.48 mmHg. Preterm births accounted for 35 percent of all births

in the hypertension group, a considerable rise from the majority of full-term deliveries in the control group (87.2%).

The majority of deliveries in the control group (96%) occurred vaginally, whereas a sizable portion of deliveries in the hypertension group (45%) occurred via lower section cesarean cut.In both groups of patients: 98% in the hypertension group and 90% in the control group, the majority of the patients delivered live babies. In the group with hypertension, 10% of births had intrauterine deaths. The fetal results are displayed in Table 3.

Parameters	Control group		Hypertensive group	
	No.	Percentage	No.	Percentage
Live Birth	98	98%	90	90%
IUD	2	2%	10	10%
Birth weight (kg)	2.82±0.43		2.33±0.84	
Mean Apgar score at birth	7.7±1.38		6.32±2.23	
Mean Apgar score at 5 minutes	9.23±1.38		8.1±2.82	
NICU admission required	3	3%	59	59%

Table 3: Fetal outcome

The control group's mean birth weight was 2.82 ± 0.43 kg, while the hypertension group's mean birth weight was 2.33 ± 0.84 kg. The control group's mean APGAR score at birth was 7.7 ± 1.38 , but the hypertension group's was 6.32 ± 2.23 . The hypertension group's mean APGAR score at five minutes was 8.1 ± 2.82 , while the control group's was 9.23 ± 1.38 . A considerable percentage (59%) of newborns born to hypertension moms needed to be admitted to the NICU.Most newborns in the control group weighed between 2.6 and 3 kg, while

most patients in the hypertension group weighed between 2 and 2.5 kg.When placenta attachment was examined, it was found that most placentas in the control group had eccentric attachments (51.2%), but most placentas in the hypertension group had marginal attachments (46.8%).The information regarding the three groups' umbilical cord insertion locations is shown. Table 4 shows that the majority of placentas in both the hypertension group (59.2%) and the control group (71.8%) had a discoid form.

Shape	Control group		Hypertensive group	
	No.	Percentage	No.	Percentage
Discoid	72	72%	59	59%
Heart	17	17%	8	8%
Oval	10	10%	17	17%
Irregular	1	1%	16	16%

The mean placental weight was found to be $541.4\pm30.62g$ in the control group and $478.4\pm30.62g$ in the hypertension group upon morphometry analysis of the placentas. In one group, the mean placental diameter was 17.87 ± 1.47 cm, whereas in the control group it was 19.87 ± 1.47 cm.

Comparing the hypertension group to the control group, there was a substantial decrease in placental area, volume, and thickness. The control group had an average of 18.46 ± 1.54 cotyledons, while the hypertension group had an average of 16.46 ± 1.54 cotyledons (Table 5).

Table 5: Placental Morphometry					
Parameters	Control group	Hypertensive group	p-value		
Mean placental weight (g)	541.4±30.62	478.4±30.62	< 0.05		
Mean placental diameter (cm)	19.87±1.47	17.87±1.47	< 0.05		
Mean placental area (cm ²)	266.24±19.75	204.77±16.89	< 0.05		
Mean placental volume (cc)	425.19±31.53	239.02±19.74	< 0.05		
Mean placental thickness (cm)	2.16±0.16	2.01±0.11	< 0.05		
Mean number of cotyledons (n)	18.46±1.54	16.46±1.54	< 0.05		

The majority of the placental weight in the hypertension group was between 451 to 500 grams, whereas it was between 500 to 550 grams in the control group (Table 6).

Placental weight (g)	Control group		Hypertensive group			
	No.	Percentage	No.	Percentage		
400-450	0		21	21%		
451-500	12	12%	47	47%		
501-550	47	47%	32	32%		
551-600	41	41%	0	0		

Table 6: Distrib	ution of cases	according to	placental	weight
------------------	----------------	--------------	-----------	--------

After calculations were made, the placental coefficient and the foetoplacental ratio were found to be 5.23 ± 0.86 and 0.19±0.02 in the control group and 4.88±1.79 and 0.17±0.06 in the hypertension group, respectively. These values were substantially lower in the hypertension group than in the control group. (Table 7)

	recopracental ratio and riacental co	
	Control group	Hypertensive group
Fetoplacental ratio	5.23±0.86	488±1.79
Placental coefficient	0.19±0.02	0.17±0.06

Table 7. Fetoplacental ratio and Placental coefficient

Gross examination of placenta revealed the features shown in table 8.

Table 8: Gross examination of placenta						
Gross Examination Finding	Status	Norm	Normotensive group		rtensive group	
_		No.	Percentage	No.	Percentage	
Calcification	• Present	36	36%	54	54%	
	• Absent	64	64%	46	46%	
Infarction	• Present	13	13%	42	42%	
	• Absent	87	87%	58	58%	
Placental cyst	• Present	6	6%	17	17%	
	• Absent	94	94%	83	83%	
Accessory placental lobe	• Present	7	7%	27	27%	
	• Absent	93	93%	73	73%	

Discussion

100 females with Normotension and 100 with hypertension were involved in the current study. Of the hypertensive group, 25% had preeclampsia, 50% had eclampsia, and 25% had gestational hypertension. 40%, 56.7, and 3.3%, respectively, for gestational hypertension, preeclampsia, and eclampsia were found in the study by Siva Sree Ranga, M.K. et al. [10].

Patients between the ages of 20 to 35 were chosen for the investigation. The age range of 20-25 years old accounted for the majority of cases (46.2%) in the control group, while the age group of 26-30 years old accounted for the majority of cases (42.4%) in the hypertension group. Similar to our study, the Sudanese study found that the majority of pregnant women with hypertensive illnesses were between the ages of 26 to 30 (36.2%) and 31 to 35 (289.9%), suggesting that pregnant women in their third and fourth decades have a higher incidence of hypertension [12]. Our findings were also comparable to the study by Siva Sree Ranga. M.K et al. maximum patients in the hypertensive group were in the age group of 25-29 years (53.33%) and in the normotensive group were in the age group of 20-24 years (50%) [10]. In the study by Kambale T et al., most cases belonged to 20-25 years age group, of which 16 cases were of mild PIH. Minimum numbers of cases were present in the age group of 30-35 years. There was only one case of eclampsia above 30 years of age. In the control group, 30 cases belonged to 20-25 age group and three cases were present in the age group of 30-35 years [11].

One of the causative causes of PIH is primigravida. 88.2% of the normotensive group in the current study was primigravida, and 11.8% was multigravida. Conversely, 20% of the females in the hypertensive groups were multigravida, and 80% of the females were primigravida. Between the two groups, there was no statistical difference. According to a study done in Kerela, 86.6% of the female participants in the hypertension group and 93.3% of those in the normotensive group were primigravidas [10]. In the Grant Medical College, Mumbai study, the primigravida group had a higher number of PIH patients (24 cases), of which 14 cases were moderate PIH and 7 and 3 cases were severe PIH and eclampsia, respectively. In the control group, 18 cases were primigravida and 13 were second gravida [11]. In the study by Kheir AEM et al about 58% of the hypertensive women were multiparous [12].

The majority of patients in both groups 90% in the hypertension group and 98.6% in the control group—had live births. In the Siva Sreeranga, M.K. et al. study, every patient in the normotensive group delivered delivery, whereas 93.3% of pregnancies in the hypertension group ended in a live birth. According to a study by Allen VM et al., women who had any kind of hypertension during pregnancy had a 1.4-fold increased risk of experiencing a stillbirth in comparison to those with normotension [14].

The control group mean birth weight in the current study was 2.82±0.43 kg, while the hypertension group's mean birth weight was 2.33±0.84 kg. In their research, Rahman LA noted that low birth weight newborns were significantly associated with pregnancy-induced hypertension and that mothers who gave birth to low birth weight babies were five times more likely to have had this condition [15].

In our investigation the control group mean APGAR score at birth was 7.7±1.38, but the hypertension group's was 6.32 ± 2.23 . The hypertension group's mean APGAR score at five minutes was 8.1±2.82, while the control group's was 9.23±1.38. One minute and five minute APGAR scores < 7 occurred in 125 (34.0%) and 55 (14.7%) newborns, respectively, in the Ghanaian study [13]. Only 6.6% of newborns in the normotensive group in the Kambale et al. trial had a low APGAR score at birth, compared to the majority of newborns in the hypertension groups [11].In the present investigation, the majority of placentas were connected marginally (46.8%) in the hypertension group and eccentric (51.2%) in the control group. This was comparable to a research by Pretorius, which found that in 42% of hypertensive cases, the placenta was marginally inserted, compared to 11.3% in hypertensive cases in a study by Londhe PS et al. [16, 17].

The majority of the placental weight in the hypertension group was between 451 to 500 grams, whereas it was between 500 to 550 grams in the control group. The majority of the placental weight in the control group in the Shevade S et al. study was between 500 to 600 g, whereas the same was between 400 to 500 g in the hypertension group [18].

The majority of the placental weight in the control group in the Patil GV et al. study was between 500 to 600 g, but the same was between 300 to 400 g in the hypertension group [19]. The placental coefficient was 0.19 ± 0.02 in the hypertensive group and 0.18 ± 0.02 in the control group in a study by Yadav SK et al.; in the current study, it was 0.19 ± 0.02 in the control group and 0.17 ± 0.06 in the hypertension group [20]. As a result of the decreased fetal weight, the hypertension group's fetoplacental ratio (FPR) and placental coefficient decreased in comparison to the control group.

Conclusion

In comparison to cases with normotension, a noteworthy influence of hypertension on placental morphometry and morphology was noted, which in turn had an effect on fetal weight. This implies that research on the placenta may provide information on the mother's and the fetus's health.

References

- 1. Udaina A, Bhagwat SS, Mehta CD. Relation between placental surface area infarction and foetal distress in pregnancy induced hypertension with its clinical relevance. J AnatSoc India 2004;53(1):27–30.
- 2. Robertson WB, Brosens I and Dixon HG. The pathological response of the vessels of the placental bed to hypertensive pregnancy. J Pathol Bacteriol 1967; 93:58192.

- 3. Norwitch ER, Chaur Dong HSU, Rapke JT. Acute complication of preeclampsia. Clinical Obstet Gynecol 2002;45(2):30829.
- 4. Sibai BM. Diagnosis and management of gestational hypertension and preeclampsia. Obstet Gynecol 2003; 102:18192.
- Browne JCM, Veall N. The maternal blood flow in normotensive and hypertensive women. J Obstet Gynaecol Br Emp 1953; 60:141–7.
- Landesman R, Douglas RG, Holze E. The bulbar conjunctival vascular bed in the toxemias of pregnancy. Am J ObstetGynecol 1954;68(1):170–3.
- Stock MK, Anderson DF, Phernetham TM, McLaughlin MK, Rankin JH. Vascular response of the maternal placental vasculature. J Dev Physiol 1980; 2:239–46.
- Dutta DC. The placenta and fetal membranes, hypertensive disorder of pregnancy. In: Textbook of Obstetrics, 4th edn. New Central Book Agency, Calcutta, 1998;28–40:234–55.
- 9. Majumdar S, Dasgupta H, Bhattacharya K, Bhattacharya A. A study of placenta in normal and hypertensive pregnancies. J AnatSoc India 2005;54(2):1–9.
- Siva Sree Ranga. M.K, et al. Morphological and histological variations of human placenta in hypertensive disorders of pregnancy. Int J Anat Res. 2017;5(1):359198. ISSN 2321-4287.
- 11. Kambale T. Placental morphology and fetal implications in pregnancies complicated by pregnancy induced hypertension. Med J DY Patil Univ. 2016; 9:3417.
- 12. Kheir AEM, et al. Neonatal Outcome in Hypertensive Disorders of Pregnancy in a Tertiary Neonatal Unit in Sudan. Journal of Medicine and Medical Research. 2014 August:2(5);5965.
- 13. Kwame AduBonsaffoh. Perinatal outcomes of hypertensive disorders in pregnancy at a tertiary hospital in Ghana. BMC Pregnancy and Childbirth 2017; 17:388.
- 14. Allen VM. The effect of hypertensive disorders in pregnancy on small for gestational age and stillbirth: a population-based study. BMC Pregnancy and Childbirth. 2004; 4:17.
- 15. Rahman LA, et al. Association between pregnancy induced hypertension and low birth weight; a population based casecontrol study. Asia Pac J Public Health. 2008;20(2):1528.
- Londhe PS, et al. Morphometric Study of Placenta and Its Correlation In Normal And Hypertensive Pregnancies. International Journal of Pharma and Bio Sciences. 2011 Oct Dec; 2(4):427437.
- Pretorius DH, Chau C, Poeltler DM, Mendoza A, Catanzarite VA, Hollenbach KA. Placental cord insertion, visualization with prenatal ultrasonography. J Ultrasound Med, 1996; 15: 58593.

- Palaskar PA, Chaudhary KR, Mayadeo NM. Foetoplacental weight relationship in normal pregnancy and preeclampsia-eclampsia: a comparative study. Bombay Hosp J. 2001; 43(3): 3613.
- 19. Patil GV, et al. A Study on Morphology of Placenta in pregnancy Induced Hypertension in Wayanad, Kerala. IJSR. 2014 July;3(7):20857.
- Yadav SK, et al. Placental Coefficient in Nepalese Population and It's Clinical Relevance. Int J Anat Res 2016;4(4):305862.