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Comparative Morphological Study Of Hypertensive And Non Hypertensive Human Placenta In Pregnancy

Tilak Raj^{1*}, Pawan Kumar Mahato², Husnuma³, Anand Kumar Singh⁴

1* Research Scholar Department of Anatomy, Index Medical College, Indore, M.P
 2 Professor Department of Anatomy, SSIMS, Bhilai Chhattisgarh
 3 Tutor Department of Anatomy, Dr. K.N. Singh Memorial Institute of Medical Sciences, Barabanki, U.P
 4 Associate Professor Department of Anatomy, Dr. K.N.Singh Memorial Institute Of Medical Sciences, Barabanki. U.P

*Corresponding Author: Tilak Raj
*PhD Scholar, Malwanchal University, E-mail: drtilak.pal@gmail.com

ABSTRACT

Introduction: Hypertensive disorders are a common complication during pregnancy. A thorough macroscopic examination of the placenta can offer significant insights into the prenatal health of both the baby and the mother.

Objectives:To compare the morphological changes in the placenta between hypertensive and normotensive pregnant mothers.

Methods: This study was conducted as an Observational Prospective Cohort Study. Detailed clinical histories were obtained, and placentas were collected from 40 hypertensive and 40 normotensive mothers who delivered in the labor room or operation theatre. Macroscopic examinations were performed, and the findings were recorded and analyzed statistically.

Results: The comparison of placental diameter, thickness, mean weight, volume, and surface area between the hypertensive and normotensive groups showed a statistically significant difference (p < 0.05).

Conclusion: Hypertensive disorders in pregnancy are reflected in the gross and microscopic findings of the placenta, which may have implications for the management of both the mother and fetus.

Keywords: Hypertensive Disorders, Morphology, Placenta, Pregnancy.

INTRODUCTION

Hypertensive disorders are common complications during pregnancy. Despite decades of intensive research, the mechanisms by which pregnancy initiates or aggravates hypertension remain unclear. A significant portion of maternal and neonatal mortality and morbidity can be directly attributed to the preeclampsia syndrome, which is characterized by new-onset hypertension accompanied by proteinuria during pregnancy [1].

The National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy classifies hypertensive disorders during pregnancy into four categories: 1) chronic hypertension, 2) preeclampsia-eclampsia, 3) preeclampsia superimposed on chronic hypertension, and 4) gestational hypertension (which includes transient hypertension of pregnancy or chronic hypertension identified in the latter half of pregnancy) [2]. A pregnant woman is considered normotensive if her blood pressure remains below 140/90 mmHg throughout the pregnancy [3].

The fetus's intrauterine existence relies heavily on a vital organ, the placenta [4]. A detailed examination of the placenta can provide significant insights into the prenatal health of both the baby and the mother. The placenta plays a crucial role in maintaining pregnancy and promoting normal fetal development. It is often considered a mirror reflecting the intrauterine environment of the fetus [5]. Research has shown that maternal utero-placental blood flow decreases in preeclampsia due to maternal vasospasm, leading to fetal hypoxia [6,7]. Near-term fetal hypoxia is relatively common and can result in fetal distress or even fetal death [8]. The reduction in maternal utero-placental blood flow, which indirectly leads to the constriction of fetal stem arteries, has been linked to the changes observed in the placentas of preeclamptic women [9]. Placentas from preeclamptic pregnancies have been found to have significantly lower total volumes of parenchyma and villous surface area compared to those from normal pregnancies of similar gestational age [10]. Minor areas of infarction are observed in about 25% of placentas from normal pregnancies, while extensive placental infarction is typically seen in placentas from preeclamptic mothers [3]. Placentas from preeclamptic pregnancies tend to be smaller on average, with more numerous and larger infarcts, often located centrally [11]. Histopathological findings such as cytotrophoblastic cellular proliferation, syncytial knot formation, and fibrin plaque formation are more prevalent in hypertensive placentas [10].

OBJECTIVES

Examining the placenta can provide valuable insights that may be crucial for the immediate and future care of both the mother and the infant. Additionally, this information can be vital in protecting the attending physician in cases of adverse maternal or fetal outcomes. It is standard medical practice to universally examine the placenta in the delivery

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room, document the findings, and submit tissue for pathological evaluation when there are abnormal appearances or specific clinical indications.

With this context in mind, the study was conducted with the following objectives:

a. To conduct a comparative analysis of these morphological changes between hypertensive and normotensive pregnant women.

MATERIALS AND METHODS

Study type: Observational study.

Study design: Prospective Cohort Study.

Study Setting, Population, and Period: The research took place over the course of one year, from March 1, 2022, to February 28, 2023. It was carried out at the Department of Anatomy and the Department of Gynaecology and Obstetrics of Index Medical College & Hospital, Indore, M.P, involving pregnant women attending these departments.

Sampling:

Selection of Cohorts: A cohort of pregnant women with hypertension, defined as having a blood pressure (BP) $\geq 140/90$ mm Hg, was selected from those attending the antenatal clinic at the Department of Obstetrics & Gynaecology, Index Medical College & Hospital. Out of the 55 hypertensive women initially selected, 40 met the inclusion and exclusion criteria at the time of delivery and in the postnatal period extending beyond 12 weeks.

A normotensive cohort, consisting of pregnant women with BP less than 140/90 mm Hg and no other illnesses, was also randomly selected from the antenatal clinic after applying the same inclusion and exclusion criteria. Of the 50 normotensive women initially chosen, 40 were traced at the time of delivery and thereafter, with their placentas collected for examination.

Inclusion Criteria: The study included pregnant women aged 20 to 40 years who attended antenatal check-ups and provided detailed histories, clinical data, consent, and cooperation. Only those with available blood and urine reports and placental specimens from full-term deliveries (37 weeks of gestation or more) were included.

Exclusion Criteria: Women younger than 20 years or older than 40 years were excluded, as well as those with other medical or surgical illnesses. Patients whose blood and urine reports were unavailable, who did not provide consent, or who were lost or missing during the follow-up period were also excluded.

Techniques in Detail: The study involved a clinical examination of cases and post-delivery analysis of the placenta as outlined below:

a. **Detailed Case History and Clinical Examination:** The last menstrual period (LMP) was recorded, and a thorough clinical examination was conducted.

Examination of the Specimen:Placental examinations were carried out following a proforma initially adopted by Benirschke and later modified by Woodly et al. [13].

• Gross Examination: The following parameters of the placenta were evaluated: dimensions, surface area, weight, volume, shape, fetal surface examination, umbilical cord insertion, amniotic membrane condition, maternal surface examination, presence of blood clots, infarcts, number of cotyledons, and examination of cut sections.

Statistical Analysis: The data were analyzed using Epi-Info software, Version 3.3.2. Statistical tests such as the Pearson Chi-square Test and Independent Samples Test were applied as necessary. A p-value of <0.05 was considered significant.

RESULT:

Table 1: Comparison of mean Blood Pressure (mmHg) between Hypertensive and Non-Hypertensive women

	Hypertensive (n=40)		Non-Hypertensive (n=40)		t	p-Value
	Mean ±SD		Mean	±SD		
Systolic Blood Pressure	136.17	8.37	105.20	6.76	18.55	0.001
Diastolic Blood Pressure	83.54	5.39	63.68	6.62	14.34	0.001

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The comparison of mean blood pressure between hypertensive and non-hypertensive women shows a highly significant difference.

Table 2. shows the comparison of mean placenta weight between hypertensive and non-hypertensive women. the mean placenta weight was significantly lower in the hypertensive group compared to non-hypertensive women.

Table 2: Comparison of mean Placenta Weight (gm) between Hypertensive and Non-Hypertensive women

	Hypertensive (n=40)		Non-Hype (n=40)	ertensive	t	p-Value
	Mean	±SD	Mean	±SD		
Placenta Weight (gm)	380.17	29.27	441.53	52.02	-7.90	< 0.001

Table 3: Comparison of frequency of Placenta Shape between Hypertensive and Non-Hypertensive women

			Hypertensive (n=40)		Non-Hypertensive (n=40)		p-Value
		n	%	n	%		
	Round	24	40.68	18	30.51	9.17	0.027
Placenta	Oval	18	30.51	33	55.93		
Shape	Discoid	15	25.42	7	11.86		
	Bidiscoid	3	5.08	1	1.69		

The comparison of placenta shape frequency between hypertensive and non-hypertensive women reveals a statistically significant difference. Moreover, the mean placenta thickness was significantly lower in the hypertensive group compared to non-hypertensive women.

Table 4: Comparison of mean Placenta Thickness between Hypertensive and Non-Hypertensive women

	Hypertensive (n=40)		Non-Hypertensive (n=40)		t	p-Value
	Mean	±SD	Mean	±SD		
Placenta Thickness (cm)	2.44	0.95	3.13	0.76	-4.39	< 0.001

Table 5 shows the comparison of mean placenta diameter between hypertensive and non-hypertensive women, the mean placenta diameter was significantly lower in the hypertensive group compared to non-hypertensive women.

Table 5: Comparison of mean Placenta diameter between Hypertensive and Non-Hypertensive women

	Hypertensive (n=40)		Non-Hypertensive (n=40)			p-Value
	Mean	±SD	Mean	±SD		
Placenta Diameter (cm)	11.80	2.33	14.42	3.02	-5.28	< 0.001

DISCUSSION

The placenta, being a fetal organ, is subjected to the same stress and strain as the fetus. Therefore, any disease affecting the mother and fetus also significantly impacts the placenta. Normally, placental morphology varies greatly during its short lifespan. The placenta, which is functionally the most critical organ during intrauterine life, is susceptible to various defects and diseases, just like other vital organs in the body. Clinical conditions such as anemia, diabetes, and hypertension can have detrimental effects on the placenta, potentially affecting the health and even the life of the fetus.

This study was conducted to examine macroscopic changes in placentas from hypertensive and normotensive mothers. While some variations were found to be very specific between the two groups, other changes were observed that could not be correlated between them. The observations can be discussed according to the following categories:

In this study, the mean birth weight of babies born to hypertensive mothers was lower than that of babies born to normotensive mothers. Udainia et al. also observed a decrease in the weight of newborns in cases of pregnancy-induced hypertension (PIH). Our findings align with these observations.

Placental Morphology: Macroscopic - The diverse variations often made it challenging to differentiate a pathological placenta from a normal one. In this study, the most common shape was round, occurring in 57.5% and 62.5% of the hypertensive and normotensive groups, respectively, with no statistically significant difference between the groups (p

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>0.05). According to Shah R. K. et al. and Shanklin DR, the shape of the placenta may be round or oval, and this bears no correlation with maternal and fetal status, which supports our observation.

The mean placental diameter was significantly lower in the hypertensive group (p < 0.05). According to Gray's Anatomy by Patricia C., the average placental diameter was 18.5 cm, which was close to our observation in the normotensive group and higher than that in the hypertensive group. The difference in mean placental thickness in our study was found to be statistically significant (p < 0.05), with lower thickness observed in the hypertensive group. Patricia C., in Gray's Anatomy, stated that the thickness of the placenta was 2.3 cm, which aligns with our observation in the normotensive group.

Placental weight is a crucial factor reflecting fetal growth. In this study, placental weight and mean placental volume werefound to be significantly lower in the hypertensive group compared to the normotensive group (p < 0.05). Majumder S. and Dasgupta H. also found that the mean weight of the placenta was significantly lower in the hypertensive group compared to the normotensive group. Damania (1989), Fox (1994), and Kalousek (1994) similarly found lower placental volumes in hypertensive groups, which is consistent with our findings. Udainia A., Bhagat SS, and Mehta CD found that the mean surface area was significantly less in cases of severe hypertension (179.14 cm2) and mild hypertension (195.98 cm2) compared to the control group (242.56 cm2). Majumder S., Dasgupta H., Damania (1989), Fox (1994), and Kalousek (1994) had similar observations.

CONCLUSION

Hypertensive disorders during pregnancy can lead to significant macroscopicchanges in the placenta, with the severity of these changes often increasing as the condition worsens. Identifying these changes may assist in the treatment of both the hypertensive mother and her child. Preventing hypertension in pregnancy could reduce the risk of fetal hypoxia and low birth weight. Additionally, this knowledge may safeguard the medical team in cases of adverse pregnancy outcomes, particularly when the patient lacks a proper history of prenatal care and presents in an emergency during delivery.

REFERENCES

- 1. Roberts JM, Pearson G, Cutler J, et al. Summary of the NHLBI Working Group on Research on Hypertension During Pregnancy. Hypertension. 2003 Mar; 41(3): 437-45. Epub 2003 Feb 10. DOI:10. 1161/01. HYP.000005 4981. 03589.E9
- 2. Barton JR, O'brien JM, et al. Mild gestational hypertension remote from term: progression and outcome. Am J Obstet Gynecol. 2001 Apr;184(5):979 83. DOI:10.1067/mob.2001.112905
- 3. Salgado SS, Pathmeswaran A. Effects of placental infarctions on the fetal outcome in pregnancies complicated by hypertension. J Coll Physicians Surg Pak. 2008 Apr;18 (4):213-6. doi: 04.2008/JCPSP. 213216.
- 4. Udainia A, Jain ML. Morphological study of placenta in pregnancy induced hypertension with its clinical relevance. Journal of the Anatomical Society of India. 2001 Jun; 50(1): 24-7.
- 5. Udainia A. Bhagwat SS, Mehta CD. Relation between placental surface area, infarction and foetal distress in pregnancy induced hypertension with its clinical relevance. J Anat. Soc. Ind 2004; 53(1): 27-30. 6. Browne JC, Veall N. The maternal placental blood flow in normotensive and hypertensive women. J ObstetGynaecol Br Emp. 1953 Apr;60(2):141-7.
- 6. Landesman R, Douglas RG, Holze E. The bulbar conjunctival vascular bed in the toxemias of pregnancy. Am J Obstet Gynecol. 1954 Jul;68(1):170-83. DOI:10.1016/0002-9378(54)90476-7
- 7. Thomson AM, Billewicz WZ, Hytten FE. The weight of the placenta in relation to birthweight. J ObstetGynaecol Br Commonw. 1969 Oct;76(10):865-72.
- 8. Stock MK, Anderson DF, Phernetton TM, et al. Vascular response of the fetal placenta to local occlusion of the maternal placental vasculature. J Dev Physiol. 1980 Oct;2(5):339-46.
- 9. Majumdar S, Dusguspta H, Bhattacharya K, Bhattcharya A. A study of placenta in normal & hypertensive pregnancies. J Anat. Soc. India 2005; 54(2): 1-9.
- 10. Wentworth P. Placental infarction and toxemia of pregnancy. Am J Obstet Gynecol. 1967 Oct 1;99(3):318-26. DOI:10.1016/s0002-9378(16)34537-9
- 11. Joseph F Yetter III. Col, MC, USA, 'American family Physician' march 1998; Vol. 57 / No. 5 Published by American Academy Of Family Physician.
- 12. Benirschk. American Journal of Obstetrics and Gynaecology .1, New York 961;84:1595.
- 13. Shah R K et al. Journal of Obstetrics and Gynecology of India 1985;35:1809.
- 14. Shanklin D R. Journal of Obstetrics and Gynecology of the British Commonwealth 1958; 11: 129.
- 15. Patricia C. Gray's Anatomy. 38th ed. New York: Churchill Livingstone,
- 16. Damania KR, Salvi VS, Ratnaparki SK, Daftari SN. The placenta in hypertensive disorder in pregnancy. J Obst and Gynaecol Ind 1989; 39: 28-31.
- 17. Fox H; The placenta in intra uterine growth retardation. In Ward RHT, Smith SK, Donnai D (Eds). Early foetal growth and development. RCOG Press, London: 1994. p. 223-235.
- 18. Kalousek DK, Langlosis S. The effects of placental and somatic chromosomal mosaicism on foetal growth. In. Ward RHT, Smith SK, Donnai (eds), Early foetal growth and development, RCOG Press; 1994. p. 245 256.

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http://www.veterinaria.org

Article received- Revised- Accepted-



- 19. Mardi K, Sharma J. Histopathological evaluation of placentas in IUGR pregnancies. Indian J PatholMicrobiol. 2003 Oct;46(4):551-4.
- 20. Salgado SS, Angunawela P, Sirisena J, De Tissera A. Villous syncytial knots in hypertensive placentae. Sri Lanka J ObstetGynaecol 2004; 26:33-7.
- 21. Fox H. Pathology of the Placenta. 2nd ed. W B: Saunders, London; 1997a
- 22. Benirschke K & Kaufmann P. 'Pathology of Human Placenta'. 2nd ed. Newyork: Springer Verlag;1990. P. 130.
- 23. Soma H, Yoshida K, Mukaida T, et al. Morphologic changes in the hypertensive placenta. ContribGynecol Obstet. 1982;9:58-75.
- 24. Jones CJP, Fox H. An ultrastructural and ultrahistochemical study of the human placenta in maternal pre-eclampsia. Placenta 1980; 1:61-76.
- 25. Damjanov I, Linder J. Andersons Pathology. 10th ed. New York: Mosby; 1906. p. 2321.
- 26. Tinney B Parker F. The placenta in Toxaemia of pregnancy. AmJObstetGynecol 1940; 39: 1000-1005.