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Morphology, Morphometry And Histopathological Changes Of Placenta Among Hypertensive And Normotensive Pregnant Mothers - A Comparative Study

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ABSTRACT:

Introduction: *The vital organ that is essential for fetal development is placenta, its integrity is essential for a healthy pregnancy. Among the various causes of maternal and perinatal morbidity & mortality, hypertensive disorders of pregnancy (HDP) affects 5-10% of pregnancies worldwide. These disorders are associated with abnormal placentation, leading to reduced uteroplacental perfusion and subsequent fetal complications.*

Materials and Methods: *A total of sixty placentas was collected from 30 normotensive and 30 hypertensive pregnant mothers of age 18-35 years at MGMCRI, Puducherry. Patients with gestational diabetes, chronic renal disorders, or fetal anomalies were excluded. Morphological features (shape, umbilical cord insertion), morphometric parameters (weight, thickness, diameter, number of cotyledons), and histopathological changes (infarcted villi, fibrinoid necrosis, syncytial knots, etc.) of the placenta was examined and statistically analysed.*

Results: *A significant difference between the two groups was observed. Placentas from hypertensive mothers were more likely to be irregular in shape with eccentric or marginal umbilical cord insertions ($p<0.001$). Morphometrically, placentas from the hypertensive group had significantly lower weight (360±75g vs. 485±60g), diameter, thickness, and number of cotyledons compared to the normotensive group ($p<0.001$). Histopathological examination of placentas from hypertensive mothers showed a higher incidence of infarcted villi, fibrinoid necrosis, and syncytial knots.*

Conclusion: *A significant association of maternal hypertension and morphology, morphometry, and histopathology of the placenta was determined. It includes reduced placental size and compromised vascular structure, reflecting impaired placental function, contributing adverse perinatal outcomes. Examination of the placenta is a valuable tool for understanding the pathophysiology of HDP and its consequences for the foetus.*

Keywords: *Placenta, Morphology, Morphometry, Hypertensive pregnant mothers, LSCS.*

1. INTRODUCTION

The placenta is a vital organ that act as the interface between the maternal and fetal systems. Placenta is the site of nutrient, gas, and waste exchange, also it acts as endocrine and immunologic roles essential for maintenance of pregnancy. Its structural and functional integrity directly influence fetal growth, well-being, and pregnancy outcomes[1]

Placental development begins with implantation, when trophoblastic cells differentiate into cytotrophoblasts and syncytiotrophoblasts. The syncytiotrophoblast invades the maternal endometrium, eroding capillaries and forming lacunae that eventually evolve into the intervillous space[2]. Through successive branching of chorionic villi, a complex vascular tree develops, maximizing the surface area for exchange. By term, the placenta is discoid, measuring approximately 15–20 cm in diameter, 2–3 cm thick, and weighing about 450–600 g. The fetal surface is smooth and glistening, while the maternal surface is dull and divided into 15–20 cotyledons[3].

Functionally, the placenta acts as a semipermeable barrier regulating exchange of oxygen, carbon dioxide, glucose, amino acids, and electrolytes[4]. It also synthesizes hormones such as human chorionic gonadotropin, progesterone, oestrogens, and placental lactogen, which maintain pregnancy and modulate maternal physiology. In addition, it protects the foetus from immune rejection through mechanisms like HLA-G expression and secretion of immunosuppressive cytokines[5]. Thus, any morphological or histological alteration can impair placental function and adversely affect fetal outcome.

1.1. HYPERTENSIVE DISORDERS OF PREGNANCY

Hypertensive disorders of pregnancy (HDP) are among the most common medical complications of gestation, affecting approximately 5–10% of pregnancies worldwide[6]. They include chronic hypertension, gestational hypertension, preeclampsia, eclampsia, and chronic hypertension with superimposed preeclampsia. Preeclampsia alone accounts for nearly 15% of maternal deaths globally and contributes substantially to perinatal morbidity and mortality[7].

The underlying mechanism of HDP involves abnormal placentation. Normally, cytotrophoblasts invade maternal spiral arteries, converting them into low-resistance, high-capacity vessels[8]. In hypertensive pregnancies, this remodelling is incomplete, leading to persistence of narrow, muscular arteries and reduced uteroplacental perfusion[9]. The resulting ischemia–reperfusion injury produces oxidative stress, endothelial dysfunction, and release of anti-angiogenic factors such as sFlt-1 and soluble endoglin. These cause vasoconstriction and increased vascular permeability, manifesting clinically as hypertension, proteinuria, and multiorgan involvement[10].

1.2. PLACENTAL CHANGES IN HYPERTENSION

Placentae from hypertensive pregnancies are typically smaller and irregular, with reduced weight, fewer cotyledons, infarctions, and areas of calcification[11]. The umbilical cord may show marginal insertion and decreased coiling[12]. Quantitatively, these placentae exhibit reduced diameter, thickness, and surface area, reflecting diminished functional capacity[13].

Histopathological changes include villous stromal fibrosis, syncytial knots, fibrinoid necrosis, peri villous fibrin deposition, and decidual vasculopathy characterized by fibrinoid degeneration and lipid-laden macrophages[14]. Such alterations compromise the exchange surface, resulting in intrauterine growth restriction, preterm birth, and fetal distress. The severity of these microscopic lesions often correlates with the degree of maternal hypertension and adverse perinatal outcome[15].

1.3. RATIONALE OF THE STUDY

The placenta provides a permanent record of maternal and fetal health during pregnancy; thus, its examination offers valuable insights into disease mechanisms and outcomes. A comparative study of the morphology, morphometry, and histopathology of placentae from hypertensive and normotensive mothers can reveal the structural basis of placental dysfunction and its relation to adverse fetal outcomes. Such analyses not only enhance understanding of the pathophysiology of hypertensive disorders but also help identify potential predictive or prognostic markers. This study is to evaluate and compare the morphological, morphometric, and histopathological features of placentae among hypertensive and normotensive pregnant mothers, thereby establishing a correlation between maternal hypertension and placental changes that influence fetal growth and pregnancy outcome.

2. AIM AND OBJECTIVES:

Aim:

To compare the histomorphological and morphometric changes of placenta between normotensive and hypertensive pregnant mothers.

Objectives:

1) To study the morphology and morphometry of placenta in normotensive and hypertensive pregnant mother.

2) To study the histopathological changes in placenta among normotensive and hypertensive pregnant mothers.

3) To compare the changes between normotensive and hypertensive placenta.

3. MATERIAL AND METHODS:

• This study was carried out in Department of Anatomy and in Department of Obstetrics & Gynaecology at MGMCRI, Puducherry. After Ethical clearance, 60 placentas were collected from Department of Obstetrics and Gynecology. Among which, 30 placentas were collected from normotensive mothers and other 30 were collected from hypertensive mothers for 12 months and they were grouped into

• GROUP I - Normotensive mothers (systolic 100- 135, diastolic 60-85 mmHg) - (n=30).

• GROUP II - Hypertensive mothers (systolic > 140, diastolic >90 mmHg) - (n=30).

3.1. Inclusion criteria:

- Pregnant mothers in the age group between 18 -35 yrs who are attending antenatal checkup at MGMCRI, Puducherry.
- Hypertensive pregnant women having BP > 140/90 mmHg
- Willingness to participate & signing consent form after detailed explanation of the study.

3.2. Exclusion criteria:

- Patient with Gestational Diabetes Mellitus
- H/o maternal diseases such chronic renal disorder, autoimmune disease, essential hypertension, thrombophilic condition.
- Evidence of fetal anomalies.

Study Type - Observational study
Study Design- Case control study
Sampling Method - Universal sampling method

4. STUDY TECHNIQUE:

4.1. Case history - Collection

- Age
- Maturity
- Mode of delivery
- Mean blood pressure

4.2. Collection of specimens – Placentas were collected, along with umbilical cord, from the labour room or OT, and subsequently labelled. Blood clots were subjected to washing and removal procedures, followed by fixation in 10% formalin solution.

4.3. Examination of Specimens:

4.3.1.Morphological parameters[16]:

- Shape – circular, irregular & oval.
- Insertion of the umbilical cord - Central, Eccentric, Marginal
- Umbilical artery and vein were also examined

4.3.2. Morphometric parameters[16]:

- Weight of the Placenta - measured by weighing scale.

Figure: 2 weight of the placenta

- Thickness of Placenta - Assessed at three locations utilizing an elongated needle. The placenta was partitioned into three equal sections by delineating two circles on its maternal surface. The thickness was measured from the centre of the central zone, the middle zone, and the peripheral zone. The average of these three measurements was determined as the thickness of the placenta[17].

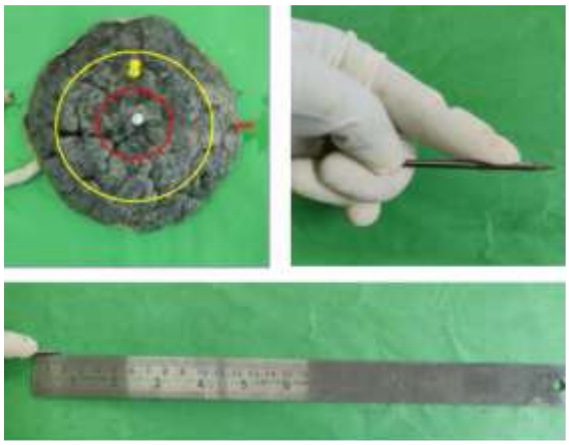

Figure: 3 Thickness measurement

• Diameter–The diameter was measured with the help of thread and measuring scale in cm. The first maximum diameter was measured by metallic scale graduated in centimeters. The second maximum diameter was taken at right angle to the first one The average of these two measurements were assessed as the diameter of the placenta[18].

Figure: 4 Diameter of the placenta

• Number of cotyledons were counted on maternal surface of placenta.

4.4. HISTOPATHOLOGY OF PLACENTA:

After macroscopic studies, Fragments of each placenta were removed from the Centre and peripheral part of the maternal and fetal surfaces. Tissues were fixed in 10% formalin and subjected to histological examination[19] these sections were stained using Hematoxylin and Eosin (H&E) stain. From each section a hundred of villi were counted and histopathological changes present in them were noted.

4.4.1.Histological Features:

1. Area of calcification 2. Infarcted villi 3. Fibrinoid necrosis of villi 4. Syncytial knots 5. cytotrophoblastic proliferation 6. Area of hyalinization.

4.4.2. Discarding the placenta:

After examining the morphometric parameters and conducting histopathological analysis, the remaining tissues were buried in the Anatomy Department burial ground. Following the analysis of variations in the placenta among normotensive and hypertensive pregnant mothers, the morphological, morphometric, and histopathological parameters were compared and the findings were systematically tabulated.

5. STATISTICAL ANALYSIS:

Quantitative data were analysed using SPSS v20.0 (IBM, Armonk, NY). Qualitative variables were presented as numbers (%) and quantitative variables as mean ± SD or median (IQR). Normality was assessed by the Shapiro–Wilk test ($p > 0.05$ indicating normality) and the Kolmogorov–Smirnov test. Associations were tested using Chi-square or Fisher's exact test for categorical variables, and independent t-test or ANOVA for numerical variables. P value < 0.05 were considered statistically significant.

6. RESULT:

Table 1 Distribution according to age

Age	Group 1	Group 2	P-value
	26.90±3.61	25.77±4.3	0.274

In this study, the mean age was compared between the two groups, as shown in Table 1. In Group 1, The age was 26.90 ± 3.61 years, while in Group 2 it was 25.77 ± 4.30 years. The difference between the two groups was not statistically significant (P value = 0.274), indicating comparable age distribution across both study groups. This demonstrated that the patients in both groups were in same reproductive age range.

Table 2: Maturity

Maturity	Group 1	Group 2	Total	P-value
Term	29(96.7%)	27 (90%)	56(93.3%)	0.250
Preterm	1 (3.3%)	3 (10%)	4 (6.7%)	
Total	30(100%)	30(100%)	30(100%)	

In his study, the gestational maturity of newborns was compared between the two groups, as shown in Table 2. Among Group 1 participants, 29 out of 30 (96.7%) deliveries were term, while only one case (3.3%) was preterm. Similarly, in Group 2, 27 out of 30 (90%) were term and 3 (10%) were preterm. Overall, 56 out of 60 (93.3%) deliveries in the study population occurred at term and only 4 (6.7%) were preterm. The difference in the term and preterm births between the two groups was not statistically significant (P value = 0.250), indicating that gestational maturity at delivery did not differ between these groups.

Table 3: Mode of delivery

Mode of delivery	Group 1	Group 2	Total	P-value
Normal vaginal delivery	20	8	28	0.004
LSCS	10	22	32	
Total	30 (100%)	30 (100%)	60 (100%)	

In this study, the mode of delivery was compared between the two groups as shown in Table 3. It showed a statistically significant difference between the two groups (P value= 0.004). Among Group 1 patients, 20 out of 30 women delivered through vaginally, whereas 10 underwent LSCS. In contrast, Group 2 demonstrated a predominance of LSCS deliveries, with 22 out of 30 undergone LSCS and only 8 had vaginal delivery. When both groups were combined, 28 of the total 60 cases had vaginal delivery and 32 delivered by LSCS. The observed difference in delivery patterns between the two groups was highly significant, suggesting that subjects in Group 2 were more likely to require LSCS intervention compared to those in Group 1.

Table 4: Mean Blood Pressure

BP (mmhg)	Group 1	Group 2	P-value
SBP	116±6	152±12	<0.001
DBP	74±5	98±8	<0.001

In this study, the blood pressure was compared between two groups as shown in Table 4. The mean systolic blood pressure (SBP) in Group 1 was 116 ± 6 mmHg, while in Group 2, it was 152 ± 12 mmHg.

Similarly, the mean diastolic blood pressure (DBP) was 74 ± 5 mmHg in Group 1 and 98 ± 8 mmHg in Group 2. The differences in both systolic and diastolic pressures between the two groups were highly significant (P < 0.001). This showed that Group 2 had higher blood pressure values compared to Group 1, confirming the presence of hypertension among participants in Group 2.

6.1. Gross: Morphological Features

Table 5: Shapes of placenta

Shapes	Group 1	Group 2	Total	P-value
Circular	18 (60%)	11 (36.7%)	29 (48.3%)	0.071
Irregular	9 (30%)	16 (53.3%)	25 (41.7%)	
Oval	3 (10%)	3 (10%)	6 (10%)	
Total	30 (100%)	30 (100%)	60 (100%)	

In this study, the shapes of Placenta were compared between 2 groups as shown in Table 5. In Group 1, the majority were circular (18 cases, 60%), followed by irregular (9 cases,30%) and oval (3 cases,10%). In contrast, Group 2 showed a higher proportion of irregular placentas (16 cases,53.3%), while circular (11 cases, 36.7%) and oval (3 cases,10%) shapes were less common. Overall, among all 60 placentas examined, circular shape was the most frequent (48.3%), followed by irregular (41.7%) and oval (10%). Though irregular placentas were more common in Group 2 compared to Group 1, this difference was not statistically significant (P value = 0.071).

Table 6: Umbilical cord insertion in the placenta

S.no	Groups	Umbilical cord insertion			Total	P- value
		Central	Eccentric	Marginal		
1	Group 1	22	8	-	30	<0.001
2	Group 2	6	14	10	30	
	Total	28	22	10	60	

In this study, the umbilical cord insertion was compared between 2 groups as shown in Table 6. The cord insertion showed a significant difference between the two groups. In Group 1, the majority of placentas had central cord insertion (22 Placentas), followed by eccentric insertion (8 Placentas), and no marginal insertions were observed. In contrast, Group 2 showed a higher proportion of abnormal insertions, with only 6 placentas showing central insertion, while 14 had eccentric and 10 had marginal insertions. Overall, out of 60 placentas examined, central insertion was found in 28, eccentric in 22, and marginal in 10. The difference of two groups were highly significant (P < 0.001), indicating that eccentric and marginal cord insertions were considerably more common in Group 2.

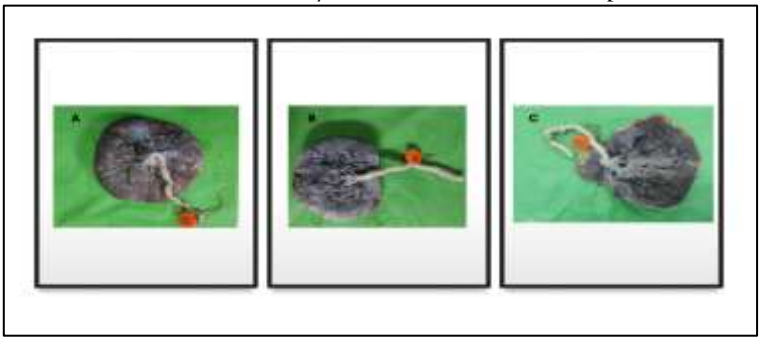

Figure : 5 shapes of placenta A) circular B) oval C) Irregular

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Figure 6: Umbilical cord insertion in Placenta A) central, B) marginal and C) eccentric

Figure 7: Placenta with two umbilical umbilical vein

Figure 8: placenta with hemorrhage arteries and one

Figure 5 – 8 depict the morphological features of the placenta. Figure 5 illustrates the different shapes of the placenta, while figure 6 shows the various sites of umbilical cord insertion. figure 7 demonstrates the presence of two umbilical arteries and one umbilical vein, which was observed in all placentas of both Group 1 and Group 2. Figure 8 shows placental hemorrhage , which was observed in placentas from hypertensive pregnancies.

6.2. Gross -Morphometric Features:

Table 7: Weight of the placenta

S.no	Parameters	Group 1	Group 2	P value
1	Weight (gms)	485±60	360±75	< 0.001

In this study, the weight of the Placenta was compared between 2 groups as shown in Table 7. The mean weight in Group 1 was 485 ± 60 grams, while in Group 2 it was significantly lower at 360 ± 75 grams. The difference of two groups were highly significant (P <0.001). These findings showed that placentas from Group 2 patients had a markedly reduced weight compared to those from Group 1.

Table 8: Thickness of the placenta

S.no	Parameters	Group 1	Group 2	P value
1	Thickness (cm)	2.3±0.4	1.8±0.3	< 0.01

In this study, The Thickness of the Placenta were compared between 2 groups as shown in Table 8. The mean thickness in Group 1 was 2.3 ± 0.4 cm, while in Group 2 it was 1.8 ± 0.3 cm. The difference of two groups were statistically significant (P < 0.01). This resulted that placentas from Group 2 patients were thinner compared to those from Group 1.

Table 9: Diameter of the placenta

S.no	Parameters	Group 1	Group 2	P value
1	Diameter (cm)	18.2±1.6	15.9±1.8	< 0.001

In this study, The Diameter of the Placenta were compared between 2 groups as shown in Table 9. The mean diameter in Group 1 was 18.2 ± 1.6 cm, while in Group 2 it was 15.9 ± 1.8 cm. The difference of two groups was highly significant (P < 0.001). This finding showed that placentas from Group 2 patients had a markedly smaller diameter compared to those from Group 1.

Table 10: Number of cotyledons in the placenta

S.no	Parameters	Group 1	Group 2	P value
1	Number of cotyledons	16.8±1.2	13.6±1.5	< 0.001

In this study, the number of cotyledons were compared between 2 groups as shown in Table 10. The mean number of cotyledons in Group 1 was 16.8 ± 1.2, whereas in Group 2 it was 13.6 ± 1.5. The difference of two groups were highly significant (P< 0.001). This showed that placentas from Group 2 patients had a lower number of cotyledons when compared to Group 1. The reduced cotyledon counts in Group 2 showed impaired villous branching and placental development, which resulted from decreased uteroplacental blood flow or vascular insufficiency associated with hypertension.

6.2.1 Morphometric features of Placenta:

This graph showed morphometric features compared between normotensive and hypertensive placenta A) Placental weight(g) B) Placental thickness(cm) C) Placental diameter(cm) D) No. of cotyledons (Figure 8(A-D)). Placentas from Group 2 were smaller and less developed than those from Group 1. The weight, thickness, diameter, and number of cotyledons were all lower in Group 2, showing that their placentas were lighter, thinner, and had fewer functional lobes.

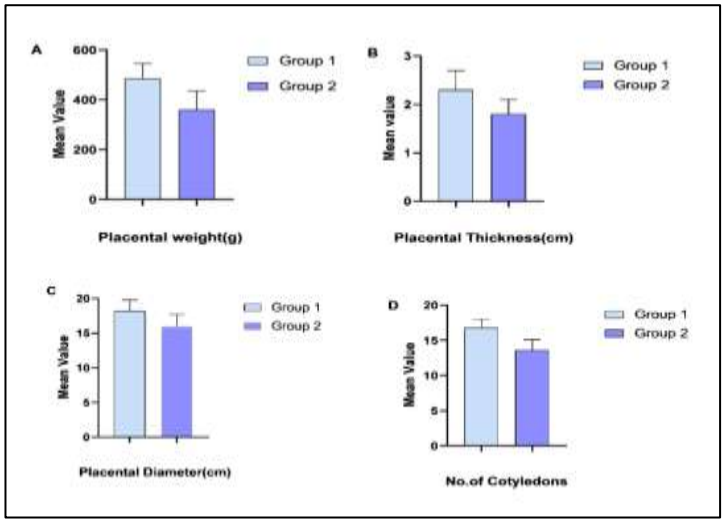

Figure 8: Morphometric features compared between normotensive and hypertensive placenta A) Placental weight(g) B) placental thickness(cm) C) Placental diameter(cm) D) No. of cotyledons.

6.3. Histopathological features of Placenta:

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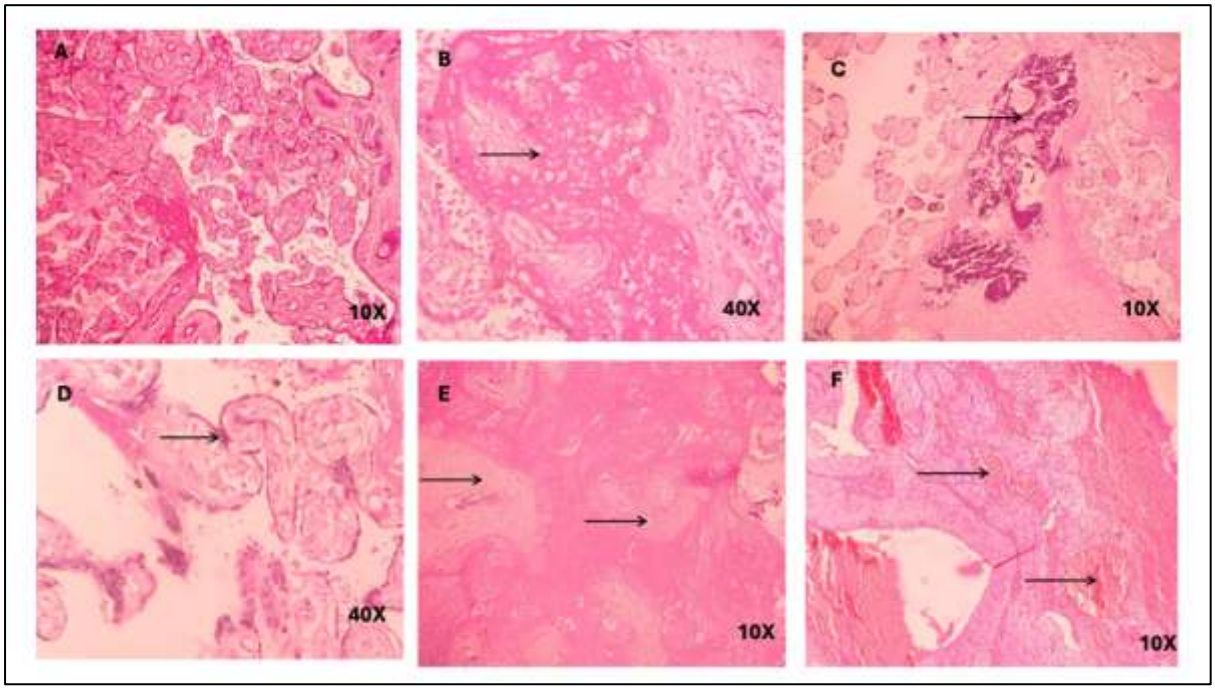

Figure 9: A) Normal placenta B) Fibrinoid necrosis of villi C) Calcification D) Syncytial knots E) Hyalinization F) Infarction.

6.3.1. Histopathological features of placenta :

This chart compared the histopathological changes observed in the placentas of Group 1 and Group 2 patients in Figure 10. It showed all abnormal features including calcification, infarction, fibrinoid necrosis of villi, syncytial knots, cytotrophoblastic proliferation, and villous hyalinization were more commonly present in Group 2 compared to Group 1. In contrast, most of these changes were absent in the placentas of Group 1. This means that placentas from hypertensive patient showed more signs of tissue damage and degeneration, indicating reduced blood flow and impaired placental function.

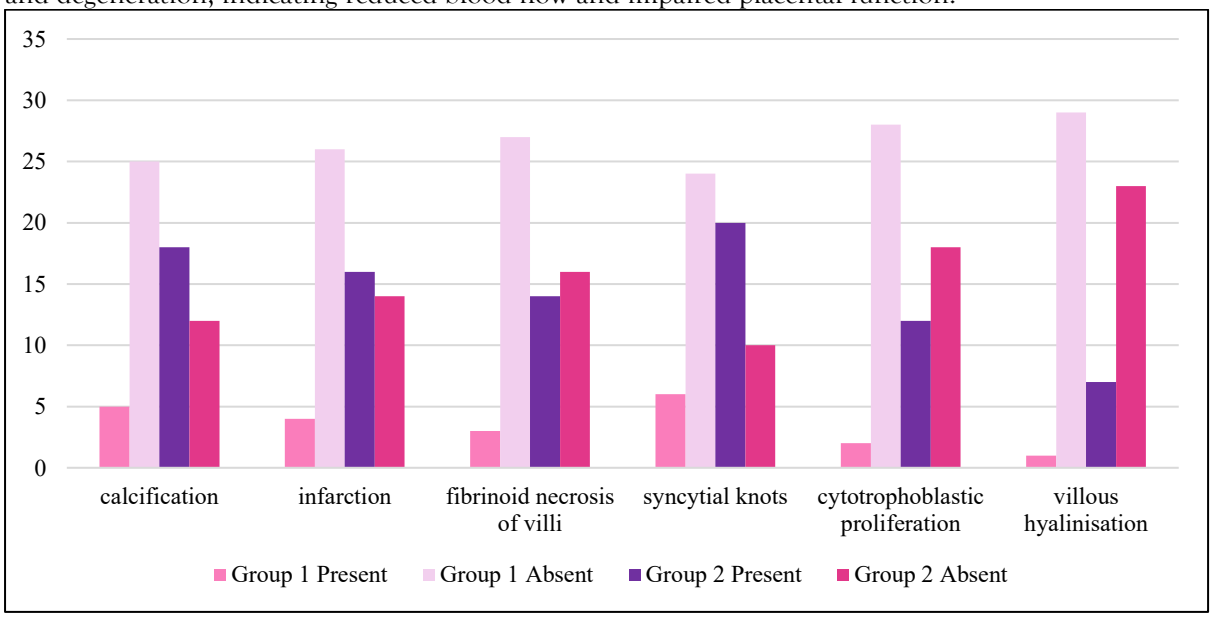

Figure 10: Comparison of histopathological features between Normotensive and Hypertensive placenta.

7. DISCUSSION:

This study showed that pregnancies affected by hypertension exhibited smaller, morphometrically compromised placenta diminished in weight, thickness, diameter, and cotyledon count with a notable

shift toward eccentric or marginal cord insertion. These changes were accompanied by an increased prevalence of ischemic–degenerative histopathological features, including infarcts, syncytial knots, fibrinoid necrosis, and calcification, collectively suggesting impaired uteroplacental perfusion with adaptive villous remodelling; these patterns were consistent with the data presented in this study results. Similar morphometric reductions had been reported previously, with Thakur et al. documenting significantly reduced placental weight and dimensions in hypertensive mothers, resembling the magnitude and direction of effect observed in the present cohort. Reductions in weight/diameter and cotyledon number in hypertensive disorders of pregnancy had likewise been reported by VR A et al. and Deshpande et al., supporting placental underdevelopment as a consistent phenotype associated with maternal hypertension. The increased occurrence of abnormal (eccentric/marginal) cord insertion observed was consistent with prior gross-pathology investigations that related hypertensive vasculopathy to altered cord implantation and reduced coiling indices, potentially compromising fetoplacental hemodynamic. Histologically, this study revealed increased syncytial knotting, villous hyalinization, and peri villous fibrin—features characteristic of preeclampsia placentopathy—which had also been documented by Kashish & Anil and by MK SS et al., supporting a hypoxia-driven pattern of villous maturation and stromal scarring. Furthermore, the severity of placental lesions had been shown by Krielessi et al. to correlate with maternal blood pressure levels, corresponding to the between-group differences in systolic/diastolic pressures and lesion prevalence in this study. Future research should have incorporated these elements to clarify pathophysiologic interactions and refine prognostication.

8. CONCLUSION:

Hypertensive disorders of pregnancy are associated with significant morphological, morphometric, and histopathological alterations of placenta. Placentas from hypertensive mothers demonstrated reduced placental dimensions and increased frequency of pathological lesions such as villous infarction, fibrinoid necrosis, and excessive syncytial knots formation compared with normotensive controls. These finding indicate impaired uteroplacental perfusion and placental insufficiency. Comprehensive placental examination may serve as a valuable adjunct for understanding disease pathophysiology, stratifying perinatal risk, and improving maternal – fetal outcome assessment in hypertensive pregnancies.

Ethical approval: The study was conducted after obtaining approval from the Institutional Human Ethics Committee (Project No : MGMCRI/Res/01/2023/50/IHEC/08).

Conflict of interest: The author declare no conflict of interest.

Author contributions:

Banu Priya P – data collection and drafting of original manuscript ; **Chandra Philip X** – conceptualization and study design; **Dr. Keerthika sri. E.S** – pathological evaluation and interpretation; **D. Suriya**– clinical data acquisition and patient management; **Arul moli R** – supervision, administrative support and critical review of manuscript, **G. Prabavathy** – methodology guidance, supervision of placental parameter measurements, and critical review of manuscript.

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