

STUDY ON PLACENTA IN PREGNANCY INDUCED HYPERTENSION –MORPHOLOGICAL CHANGES AND SPECIAL STAINS – A CASE CONTROL STUDY

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ABSTRACT

Background: Pregnancy-induced hypertension (PIH) can reduce uteroplacental blood flow and produce structural changes in the placenta. Examination of these changes may help in understanding adverse fetal outcomes. **Aim:** To compare the gross and histopathological features of placentas from pregnancies complicated by PIH with those from normotensive term pregnancies. **Materials and Methods:** This prospective comparative study included 60 placentas collected between November 2022 and April 2024. Thirty placentas were from women with PIH, including 17 with pre-eclampsia and 13 with gestational hypertension, and 30 were from normotensive term pregnancies. Placental morphometric parameters and gross abnormalities were recorded. Histological sections were stained with haematoxylin and eosin. Periodic acid–Schiff stain was used to demonstrate trophoblastic basement membrane thickening. The findings were analysed using the chi-square test and unpaired Student's t-test. **Results:** Mean neonatal birth weight was significantly lower in the PIH group than in the control group (2308.7 ± 537.71 g versus 2858.3 ± 307.32 g; $p < 0.001$). Placental weight, diameter and number of cotyledons were also significantly reduced in the PIH group. Abnormal cord insertion and placental infarction involving more than 5% of the parenchyma were more frequent in PIH. Increased syncytial knotting, villous fibrinoid necrosis, trophoblastic basement membrane thickening and hyalinised villi were significantly more common in PIH placentas. **Conclusion:** PIH was associated with reduced placental growth and increased ischaemic and degenerative changes. These alterations may contribute to lower fetal birth weight. Placental examination, together with PAS staining when required, can help in demonstrating these changes.

INTRODUCTION

The placenta is a vital organ that acts as a diary of intrauterine life. It provides an accurate record of the infant's prenatal experience. It is a fetomaternal organ directly related to the growth and development of the fetus in the uterus. The placenta is essential for the exchange of respiratory gases, nutrients and waste products between the mother and fetus. It undergoes continuous changes in weight, volume, structure, shape and function throughout gestation to support prenatal life.^[1,2] Thus, a thorough examination of the placenta, both in utero and after delivery, may provide an insight into the prenatal health of the mother and baby.

Hypertensive disorders of pregnancy are common obstetric complications and remain important causes of maternal and perinatal morbidity and mortality,

particularly in developing countries. Gestational hypertension is characterised by the new onset of hypertension after 20 weeks of gestation without proteinuria or other features of pre-eclampsia. Pre-eclampsia is diagnosed when hypertension is associated with proteinuria or evidence of maternal organ dysfunction.^[3] These conditions can adversely affect placental morphology and may result in fetal growth restriction and other adverse perinatal outcomes.

According to the Sample Registration System report for 2021–2023, the maternal mortality ratio was 88 per 100,000 live births in India and 35 per 100,000 live births in Tamil Nadu.^[4] Hypertensive disorders of pregnancy continue to contribute significantly to maternal deaths worldwide.^[5] In developing countries, the condition may remain undetected until major complications occur. Maternal and placental

factors play an important role in the pathophysiology of pre-eclampsia. Abnormal placental development during early pregnancy leads to reduced placental perfusion, hypoxia and ischaemia. This results in the release of antiangiogenic factors into the maternal circulation and alteration of maternal endothelial function, leading to hypertension and other manifestations of the disease.^[6]

Incomplete and shallow trophoblastic invasion of the maternal spiral arteries is an important feature of abnormal placental development. This results in the persistence of muscular and elastic tissues within the walls of the spiral arteries.^[7] These vessels remain responsive to vasomotor influences, resulting in a high-resistance and low-flow uteroplacental circulation. The placenta therefore acts as a mirror of maternal and fetal health and reflects the changes occurring in hypertensive disorders of pregnancy.

Placental examination has gained importance among pathologists in understanding the pathogenesis of pre-eclampsia and other pregnancy-related disorders. A systematic gross and histopathological examination of the placenta can provide useful information regarding maternal vascular malperfusion and fetal well-being.⁸ Placental examination is a simple and non-invasive procedure. The present study was undertaken to assess the morphological and histopathological changes in placentas from pregnancies complicated by pregnancy-induced hypertension and to compare them with placentas from normotensive pregnancies.

Aim and Objectives: To observe and study the various gross morphological changes and histopathological changes in placentas of pregnancy induced hypertension mothers and its comparison with normal term placentas. To draw necessary conclusions from the study and analyse the results with available various studies in the literature.

MATERIALS AND METHODS

Study design: This prospective comparative study was conducted in the Department of Pathology in collaboration with the Department of Obstetrics and Gynaecology, Government Mohan Kumaramangalam Medical College and Hospital, Salem. The study was carried out over 18 months, from November 2022 to April 2024.

Study population: A total of 60 placentas were studied. The cases were divided into two groups:

- Control group: 30 placentas from women with uncomplicated pregnancies and normal blood pressure, without proteinuria.
- PIH group: 30 placentas from women diagnosed with gestational hypertension or pre-eclampsia after 20 weeks of gestation.

The participants were selected from women admitted to the Department of Obstetrics and Gynaecology during the study period.

Diagnostic criteria

Pregnancy-induced hypertension was defined as a blood pressure of $\geq 140/90$ mmHg, recorded on at least two occasions six hours or more apart after 20 weeks of gestation, with or without proteinuria. Relevant clinical and obstetric details were obtained from the case records and entered in a predesigned proforma. The birth weight of the neonate was also recorded.

Inclusion Criteria

Control group

Women with uncomplicated term pregnancies, normal blood pressure and no proteinuria were included.

PIH group

Women beyond 20 weeks of gestation with a blood pressure of $\geq 140/90$ mmHg, with or without proteinuria, were included.

Exclusion Criteria

Women with the following conditions were excluded:

- Hypertension diagnosed before pregnancy
- Hypertension secondary to another underlying disorder
- Proteinuria due to renal or other non-obstetric causes
- Multiple gestation

Collection and gross examination of the placenta

The placenta, together with the attached membranes and umbilical cord, was collected immediately after vaginal or caesarean delivery. Adherent blood clots were removed by gentle washing under running tap water. The placenta was examined in the fresh state and handled carefully to avoid producing artificial tears or lacerations.

The shape and weight of the placenta were recorded. The fetal surface was examined for its colour, opacity and subchorionic fibrin deposition. The maternal surface was assessed for completeness, number of cotyledons, adherent blood clots, calcification and infarction. The extent and distribution of visible infarcts and calcifications were noted. The extraplacental membranes were examined for colour, transparency, completeness and the presence of clots or other gross abnormalities.

Tissue sections of 3-5 μ m thickness were cut from paraffin embedded blocks and stained by conventional Haematoxylin and Eosin stain. According to the findings observed, special staining techniques like periodic acid Schiff (PAS) for basement membrane and fibrinoid necrosis were employed. The slides were examined under light microscopy and the different histological features were quantified. For quantification, one hundred terminal villi from each slide were counted and mean calculated. The mean number of villi showing syncytial knots, fibrinoid necrosis, perivillous fibrin deposits, basement membrane thickening were noted.

Statistical Analysis: All the data were arranged in excel workbook under appropriate headings. The statistical analysis was done using SPSS Statistical Software 23.0 version and the qualitative variables

were analysed by Chi Square test and the quantitative variables by unpaired Student t Test. The probability value (p value) of less than 0.05 was taken as statistically significant and a value less than 0.001 as highly significant.

RESULTS

A total of 60 placentas were studied. Thirty placentas were obtained from normotensive term pregnancies and formed the control group. The remaining 30 placentas were from pregnancies complicated by pregnancy-induced hypertension (PIH). The PIH group included 17 cases of pre-eclampsia and 13 cases of gestational hypertension.

The maternal and neonatal characteristics are shown in Table 1. Most mothers in both groups were 21-25 years old. Mean maternal age was higher in the PIH group ($p = 0.042$), although the distribution of the predefined age groups was not significantly different. Parity was also comparable between the groups. Mean neonatal birth weight was lower in the PIH

group than in the control group (2308.7 ± 537.71 g versus 2858.3 ± 307.32 g; $p < 0.001$).

The gross placental findings are given in Table 2. Mean placental weight, placental diameter and number of cotyledons were lower in the PIH group. Placental thickness did not show a significant difference. Abnormal cord insertion was more frequent in the PIH group ($p = 0.008$). Infarction involving more than 5% of the placental parenchyma was present in 11 PIH placentas and was not seen in the control group ($p < 0.001$). The frequency of calcification was not significantly different between the groups.

The histopathological findings are presented in Table 3. Increased syncytial knotting, villous fibrinoid necrosis and trophoblastic basement membrane thickening were more frequent in the PIH group (all $p < 0.001$). Periodic acid-Schiff staining highlighted the basement membrane thickening. Hyalinised villi were also more frequent in the PIH group ($p = 0.007$). Decidual vasculopathy was identified in four PIH placentas and in none of the control placentas; however, this difference was not statistically significant by Fisher's exact test ($p = 0.112$).

Table 1: Maternal and neonatal characteristics

Characteristic	Category or statistic	Control (n = 30)	PIH (n = 30)	p value
Maternal age, years	Mean $\pm$ SD	23.20 $\pm$ 3.418	25.53 $\pm$ 5.077	0.042
Age group	≤ 20 years	8 (26.7)	6 (20.0)	0.277
	21-25 years	15 (50.0)	12 (40.0)	
	26-30 years	6 (20.0)	6 (20.0)	
	> 30 years	1 (3.3)	6 (20.0)	
Parity	Primigravida	16 (53.3)	19 (63.3)	0.432
	Multigravida	14 (46.7)	11 (36.7)	
Neonatal birth weight, g	Mean $\pm$ SD	2858.3 $\pm$ 307.32	2308.7 $\pm$ 537.71	< 0.001

Table 2: Gross placental and morphometric findings

Parameter	Category or statistic	Control (n = 30)	PIH (n = 30)	p value
Placental weight, g	Mean $\pm$ SD	482.33 $\pm$ 79.87	428.03 $\pm$ 88.20	0.015
Fetal-placental weight ratio	Ratio	5.92:1	5.39:1	—
Shape	Round	17 (56.7)	12 (40.0)	0.270
	Oval	13 (43.3)	16 (53.3)	
	Irregular	0	2 (6.7)	
Cord insertion	Central	25 (83.3)	14 (46.7)	0.008
	Eccentric	5 (16.7)	14 (46.7)	
	Marginal	0	2 (6.7)	
Placental diameter, cm	Mean $\pm$ SD	16.066 $\pm$ 2.300	15.033 $\pm$ 1.401	0.041
Placental thickness, cm	Mean $\pm$ SD	2.436 $\pm$ 0.436	2.215 $\pm$ 0.490	0.070
Number of cotyledons	Mean $\pm$ SD	15.10 $\pm$ 2.40	13.33 $\pm$ 2.07	0.003
Calcification	Present	11 (36.7)	14 (46.7)	0.432
Infarction $> 5\%$	Present	0	11 (36.7)	< 0.001

Table 3: Histopathological findings

Finding	Assessment	rol (n = 30)	PIH (n = 30)	p value
Syncytial knots	$> 30\%$ of villi, n (%)	2 (6.7)	22 (73.3)	$< 0.001^*$
	Mean villi $\pm$ SD	16.40 $\pm$ 7.92	35.73 $\pm$ 15.035	$< 0.001^*$
Fibrinoid necrosis	$> 3\%$ of villi, n (%)	8 (26.7)	28 (93.3)	$< 0.001^*$
	Mean villi $\pm$ SD	4.23 $\pm$ 2.285	12.70 $\pm$ 4.340	$< 0.001^*$
Trophoblastic BM thickening	$> 3\%$ of villi, n (%)	2 (6.7)	25 (83.3)	$< 0.001^*$
	Mean villi $\pm$ SD	1.33 $\pm$ 1.184	10.03 $\pm$ 6.430	$< 0.001^*$
Hyalinised villi	Present, n (%)	3 (10.0)	12 (40.0)	0.007^*
Decidual vasculopathy	Present, n (%)	0	4 (13.3)	0.112

*Statistically significant. Values are presented as mean $\pm$ standard deviation or n (%), as appropriate. Continuous variables were compared using the independent-samples t-test. Categorical variables were compared using Pearson's chi-square test or an

exact test when expected cell counts were small. BM, basement membrane; PIH, pregnancy-induced hypertension. An em dash indicates that a statistical comparison was not performed.

Table 4: Comparison of fetal birth weight, placental weight and fetoplacental ratio across studies

Parameter	Study	Control	PIH
Mean birth weight (g)	Udainia et al. [14]	2640	2280
	Majumdar et al. [15]	2800	2040
	Ranga et al. [16]	2900	2500
	Present study	2858	2308
Mean placental weight (g)	Udainia et al. [14]	495	405.6
	Majumdar et al. [15]	485.8	399.1
	Iqbal et al. [18]	489.1	405.2
	Present study	482.33	428.03
Fetoplacental ratio	Majumdar et al. [15]	5.89:1	6.23:1
	Iqbal et al. [18]	5.68:1	5.38:1
	Present study	5.92:1	5.39:1

Table 5: Comparison of placental morphometric parameters across studies

Parameter	Study	Control	PIH
Mean diameter (cm)	Kishwara et al. [20]	18.80	16.08
	Porwal et al. [22]	19.13	16.97
	Singh et al. [24]	18.40	15.91
	Present study	16.066	15.033
Mean thickness (cm)	Kishwara et al. [20]	1.59	1.51
	Singh et al. [24]	2.77	2.39
	Keche et al. [25]	1.96	1.57
	Present study	2.43	2.21
Mean number of cotyledons	Kishwara et al. [20]	15.7	14.3
	Singh et al. [24]	16.2	10.02
	Manjunatha et al. [12]	19	18.2
	Present study	15	13

Table 6: Comparison of gross placental lesions across studies

Study	Calcification control (%)	Calcification PIH (%)	Infarction control (%)	Infarction PIH (%)
Porwal et al. [22]	46.66	66.66	6.6	40
Manjunatha et al. [12]	6.5	14.8	0	29.5
Present study	36.7	46.7	0	36.7

Table 7: Comparison of microscopic placental lesions across studies

Lesion	Study	Control (%)	PIH (%)
Syncytial knots (>30% villi)	Sreechithra Kartha et al. [26]	16	32
	Porwal et al. [22]	6.67	98.33
	Ranga et al. [16]	23.3	63.3
	Present study	6.66	73.33
Fibrinoid necrosis (>3% villi)	Sreechithra Kartha et al. [26]	2	19
	Porwal et al. [22]	26.66	36.67
	Ranga et al. [16]	10	63.3
	Present study	26.66	100
Basement membrane thickening	Narasimha et al. [28]	0	49.25
	Manjunatha et al. [12]	0	50.8
	Porwal et al. [22]	0	66.67
	Present study	6.67	83.33
Hyalinised villi	Motwani et al. [30]	13.33	46.66
	Sreechithra Kartha et al. [26]	14	27
	Present study	10	40
Decidual vasculopathy	Sreechithra Kartha et al. [26]	4	16
	Kim et al. [31]	0.4	10.2
	Present study	0	13.3



Figure 1: A-F: A - Round placenta with central insertion of cord, B - Oval placenta with eccentric insertion of cord, C- Irregular placenta with eccentric insertion of cord; D - Marginal insertion of cord; E - Cut section showing foci of infarction; F - Cut section showing foci of calcification

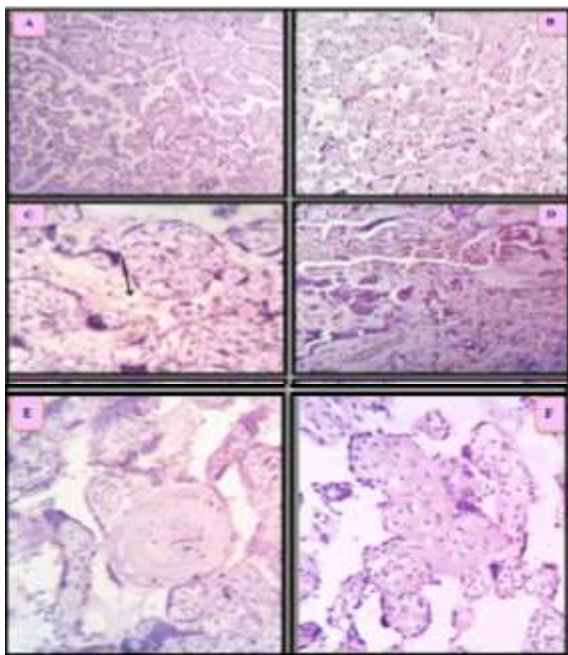


Figure 2: A-F: A - Villi from normal mother (H&E, 10X), B - Villi from PIH mother (H&E, 10X), C- Villous showing syncytial knots (H&E, 40X); D - Areas of calcification (H&E, 10X); E - Villous showing fibrinoid necrosis (H&E, 40X); F - Villous showing hyalinized villi (H&E, 40X)

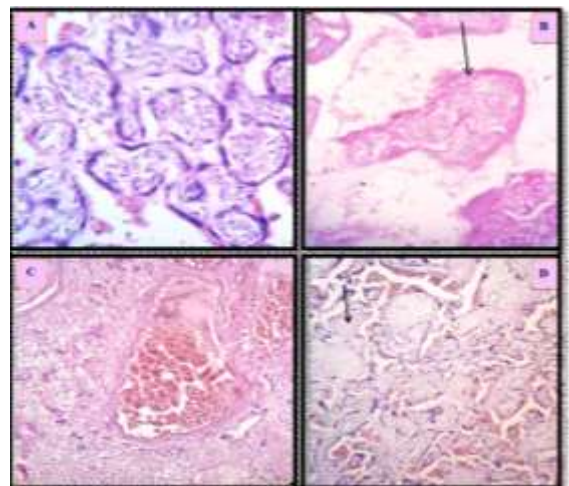


Figure 3: A-F: A - Villous showing basement membrane thickening (H&E, 40X); B - Villous showing basement membrane thickening (PAS, 40X); C- Villous showing decidual vasculopathy (H&E, 10X); D - Villous showing perivillous fibrin (H&E, 10X)

DISCUSSION

The placenta is a fetal organ with an essential role in fetal growth and is exposed to the same stresses that affect the fetus. Maternal and fetal disease may therefore produce significant placental changes. Hypertensive disorders of pregnancy are an important cause of maternal and fetal morbidity and mortality and can alter both the gross and microscopic morphology of the placenta.

The placenta is often discarded after delivery without adequate examination. Careful gross and microscopic assessment, however, contributes to understanding the pathogenesis and consequences of hypertensive disorders of pregnancy. Fox emphasized that placental pathology is largely quantitative: many lesions may occur in normal placentas, but they become clinically important when their extent exceeds the expected range and affects fetal outcome.^[9] As systematic quantification of placental changes in pregnancy-induced hypertension (PIH) remains limited, the present study compared placentas from hypertensive and normotensive pregnancies.

The PIH group included 17 mothers with pre-eclampsia (56.7%) and 13 with gestational hypertension (43.3%). No case of eclampsia was recorded, possibly reflecting timely obstetric care.

Maternal characteristics

Maternal age ranged from 18 to 36 years in both groups, with most mothers aged 21-25 years. The mean age was 23.20 $\pm$ 3.418 years in the control group and 25.53 $\pm$ 5.077 years in the PIH group. Devi Sankar et al. reported comparable mean ages of 24.36 $\pm$ 3.9 and 26.18 $\pm$ 4.41 years, respectively.^[10] In the present study, 6 of 30 women with PIH were older than 30 years. Duckitt et al. also identified maternal age above 30 years as a risk factor for PIH and pre-eclampsia.^[11]

The PIH group comprised 19 primigravidas (63.3%), including three elderly primigravidas, and 11 multigravidas (36.7%). The control group included 16 primigravidas (53.3%) and 14 multigravidas (46.7%). Manjunatha et al. and Jain et al. similarly found a greater proportion of primigravida's among mothers with PIH.^[12,13]

Fetal birth weight and placental weight

The mean birth weight was 2858.3 g in the control group and 2308.7 g in the PIH group, and the reduction in the PIH group was statistically in agreement with the findings of Udainia et al,^[14] Majumdar et al,^[15] Ranga et al.^[16] Rath attributed low birth weight in PIH to altered intracotyledonous vascular arrangement.^[17] Reduced placental blood flow, low oxygen consumption, poor glucose utilization and impaired glycolysis may further restrict nutrient synthesis and fetal growth.

Mean placental weight was lower in the PIH group than in the control group (428.03 g versus 482.33 g), in agreement with the findings of Udainia et al.,^[14] Majumdar et al,^[15] Das et al,^[18] and Iqbal B et al,^[19] The fetoplacental ratio was also lower in the PIH group (5.39:1 versus 5.92:1). Fox described compensatory placental hypertrophy in response to chronic ischaemia and hypoxia; when accompanied by reduced fetal weight, this may lower the fetoplacental ratio.^[9] These comparisons are summarized in [Table 4].

Gross placental morphology

Round, oval and irregular placentas accounted for 40%, 53.3% and 6.7% of the PIH group, respectively, compared with 56.7% round and 43.3% oval placentas in the control group. No irregular placenta was identified in the control group. Kishwara et al.^[20] also found more oval and irregular placentas in PIH, whereas Dadhich et al,^[21] reported predominantly round placentas.

Eccentric and marginal cord insertions were more frequent in hypertensive placentas, consistent with studies of Udainia et al,^[14] Majumdar et al,^[14] and Fox.^[9] In marginal insertion, altered intracotyledonous vascular arrangement may cause unequal distribution of placental blood flow and increase fetal risk.

Mean placental diameter was significantly lower in the PIH group (15.03 cm) than in the control group (16.06 cm). Similar reductions were reported by Kishwara et al,^[20] Porwal et al,^[22] and Teasdale et al.^[23] This finding may reflect reduced placental size secondary to placental insufficiency. Mean placental thickness was also lower in the PIH group (2.215 cm versus 2.436 cm), although the difference was not statistically significant. Kishwara et al.,^[20] and Das et al,^[18] reported a similar non-significant reduction, while Singh et al^[24] Ranga et al,^[16] and Keche et al,^[16] found a significant reduction. [Table 5]

The mean number of cotyledons was significantly lower in the PIH group than in the control group (13 versus 15). Kishwara et al,^[20] and Ranga et al,^[16] reported comparable findings [5,99]. Majumdar et al,^[15] and Manjunatha et al,^[12] also found fewer

cotyledons, although their differences were not statistically significant. Excess fibrin deposition with obliteration of intercotyledonous septa has been proposed as a possible explanation. Comparative morphometric data are shown in [Table 5].

Gross placental lesions

Placental calcification was present in 46.7% of PIH placentas and 36.7% of controls. Although more frequent in PIH, the difference was not statistically significant, as also reported by Sreechithra Kartha et al.^[26] Manjunatha et al,^[12] and Porwal et al,^[22] found a significant increase in PIH, while Mohan et al. found equal frequencies in hypertensive and normotensive pregnancies. Navbir et al. described an association between calcification and primigravidity, and other studies have related placental calcification to maternal serum calcium levels.^[27]

Infarction involving more than 5% of the placental parenchyma is considered clinically significant. Small infarcts may occur in uncomplicated pregnancies; Fox reported infarction in 25% of normal term placentas^[9] In the present study, infarction involving more than 5% of the parenchyma was found in 11 PIH placentas (36.7%) and in none of the controls, a statistically significant difference. Gross lesions across studies are compared in [Table 6].

Placental histomorphology

Histomorphological features were assessed quantitatively in both groups. Syncytial knots in more than 30% of villi, fibrinoid necrosis in more than 3% of villi and trophoblastic basement membrane thickening in more than 3% of villi were considered abnormal. Hyalinised villi and decidual vasculopathy were also recorded.

Increased syncytial knots were present in 73.33% of PIH placentas and 6.66% of controls. This was consistent with studies by Narasimha et al,^[28] Navbir et al,^[27] Sreechithra Kartha et al,^[26] Porwal et al,^[22] and Ranga et al,^[16,24,95-99] Pathiraja et al. cautioned that nuclear aggregates may mimic syncytial knots; true knots show densely condensed chromatin.^[29] Generalized syncytial knot formation has been related to reduced placental perfusion and altered placental morphometry.

Fibrinoid necrosis appears as a nodular mass of homogeneous eosinophilic material within villi and is also termed intravillous fibrinoid. Involvement of more than 3% of villi is considered abnormal. It was present in all PIH placentas in the present study and was significantly more frequent than in controls. Sreechithra Kartha et al,^[26] Porwal et al,^[22] and Ranga et al,^[16] reported PIH frequencies of 19%, 36.67% and 63.3%, respectively. Peri villous fibrin, in contrast, may increase with gestational maturity. Extensive deposits can resemble infarction, but villi within peri villous fibrin retain nuclear detail, whereas infarcted villi lose cellular detail and become mummified.

Trophoblastic basement membrane thickening was found in 83.33% of PIH placentas and 6.67% of controls. The thickening may result from deposition

of basement membrane proteins by proliferating cytotrophoblastic cells in response to placental ischaemia. The finding agrees with reports by Narasimha et al,^[28] Navbir et al,^[27] Manjunatha et al,^[12] and Porwal et al.^[22] Hyalinised villi with villous hypovascularity were identified in 40% of PIH placentas and 10% of controls. Similar observations were made by Motwani et al,^[30] Sreechithra Kartha et al,^[26] Ranga et al,^[16] and Majumdar et al,^[15] Reduced villous vascularity impairs nutrient transfer to the fetus.

Decidual vasculopathy, characterized by fibrinoid necrosis and/or acute atherosclerosis of uteroplacental vessels, was identified in 13.3% of PIH placentas and in none of the controls, in agreement with Sreechithra Kartha et al,^[26] and Kim et al,^[31] Acute atherosclerosis occurs in spiral arteries that fail to undergo normal physiological remodelling. Chan et al. found decidual vasculopathy more often in PIH and pre-eclampsia, followed by chronic hypertension and fetal growth restriction, and noted that some lesions were confined to basal plate sections. In the present study, the lesions were also identified in the basal plate. All cases with decidual vasculopathy had extensive infarction. Stevens et al,^[32] likewise reported an association between the number of affected vessels, infarction and accelerated villous maturity. Kim et al., associated acute atherosclerosis with small-for-gestational-age neonates, a finding also observed in the present study.^[31] [Table 7].

Small mature villi were present in 16.6% of PIH placentas, compared with 82.4% in the study by Pathiraja et al,^[29] Chorionitis was found in 6.67% of PIH placentas. Manjunatha et al,^[12] reported chorionitis in 54.1% and also observed chorioamnionitis and squamous metaplasia, neither of which was found in the present study. Comparative microscopic findings are summarized in [Table 7].

Overall, fetal birth weight, placental weight, fetoplacental ratio, placental diameter and number of cotyledons were lower in the PIH group. Placental thickness was also reduced, but the difference was not statistically significant. PIH placentas showed significantly higher frequencies of syncytial knots, fibrinoid necrosis, trophoblastic basement membrane thickening, hyalinised villi and decidual vasculopathy. These findings support the role of placental examination in documenting reduced placental perfusion and its structural consequences in hypertensive pregnancies.

CONCLUSION

Placentas from pregnancies complicated by PIH showed reduced placental weight, diameter and number of cotyledons, along with increased infarction, syncytial knotting, fibrinoid necrosis and trophoblastic basement membrane thickening. PAS stain helped in the clear demonstration of basement membrane thickening. These findings indicate that

PIH is associated with significant placental changes that may contribute to reduced fetal birth weight.

In the present study, the placental morphometric parameters were all reduced in the test group which was statistically significant ($p < 0.05$) except for placental thickness which was not statistically significant. The histopathological changes in the PIH group showed statistically significant increase in the incidence of syncytial knots, fibrinoid necrosis, thickened basement membrane and hyalinised villi.

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