Section: Anatomy

Original Research Article

MICROSCOPIC STUDIES OF HUMAN PLACENTA IN PREGNANCY INDUCED HYPERTENSIVE PATIENTS

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ABSTRACT

Background: Pregnancy-induced hypertension (PIH) significantly alters placental structure and function, posing serious risks to both maternal and foetal health. PIH remains a major contributor to perinatal morbidity and mortality, especially in low-resource settings like Eastern Uttar Pradesh, where region-specific data is scarce. Understanding microscopic placental changes associated with PIH can offer critical insights into disease pathophysiology and inform clinical management strategies. This study aimed to analyse histopathological alterations in placentas from PIH-affected pregnancies and compare them with those from normotensive pregnancies, focusing on their correlation with adverse foetal outcomes. **Materials and Methods:** A total of 100 placentas—50 from normal pregnancies and 50 from PIH cases—were collected post-delivery at B.R.D. Medical College, Gorakhpur. Samples were fixed, sectioned, and stained with haematoxylin and eosin for microscopic examination. Histopathological parameters included fibrin deposition, fibrinoid necrosis, syncytial knots, cytotrophoblastic and endothelial proliferation, and calcification. Data were statistically analysed using SPSS Version 26, with significance set at $p < 0.05$. **Result:** PIH placentas showed a significant increase in fibrin deposition (100%), fibrinoid necrosis (68%), syncytial knots (80%), cytotrophoblastic proliferation (100%), and endothelial proliferation (88%) compared to controls. Calcification was also more prevalent in PIH cases (68% vs. 52%). These findings indicate compromised placental perfusion, hypoxia-induced cellular responses, and vascular pathology, all of which contribute to placental insufficiency and adverse foetal outcomes such as intrauterine growth restriction and preterm birth. **Conclusion:** The study highlights distinct histopathological changes in PIH placentas, reinforcing their role as markers of placental dysfunction. These alterations underline the importance of early detection and management of hypertensive disorders during pregnancy to improve maternal and foetal outcomes, particularly in underserved regions.

INTRODUCTION

The placenta is essential for nutrient and oxygen exchange between mother and foetus. Pregnancy-induced hypertension (PIH) disrupts placental structure and function, leading to adverse maternal and foetal outcomes. Studying these placental changes helps understand PIH's impact and guide clinical management.

According to WHO (2023), hypertensive disorders affect about 10% of pregnancies worldwide, causing 14% of maternal deaths annually. Pre-eclampsia and eclampsia alone lead to ~70,000 maternal and 500,000 foetal deaths each year, especially in Sub-Saharan Africa and South Asia. In India, 5–10% of pregnancies are affected, with PIH contributing to 20% of maternal deaths.[1] Poor antenatal care and healthcare infrastructure increase the risk, particularly in rural areas.

PIH is defined as new-onset hypertension after 20 weeks of gestation. It includes gestational hypertension, pre-eclampsia, eclampsia, and chronic

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hypertension.[2] These conditions increase the risk of IUGR, preterm birth, organ damage, and placental complications.[3] While hypertensive disorders in pregnancy are well studied, regional data especially from Eastern Uttar Pradesh is limited. Exploring placental changes in this population can improve understanding and help develop targeted interventions.[2]

The term placenta is discoid, 400–600g in weight, and 15–20 cm in diameter. It has maternal and foetal surfaces, with chorionic villi playing a key role in exchange. Histologically, chorionic villi are lined by syncytiotrophoblasts and cytotrophoblasts.[4]

In PIH, the placenta undergoes notable changes that impair its function. Anatomically, it becomes smaller due to poor blood supply. Inadequate spiral artery remodelling leads to high vascular resistance and reduced perfusion. Physiologically, poor trophoblastic invasion causes chronic hypoxia, triggering inflammation, oxidative stress, endothelial damage, and vasoconstriction.[5] This hampers oxygen and nutrient exchange, contributing to foetal growth restriction.[2]

Pathologically, the placenta shows signs of stress such as villous ischemia, infarctions, fibrinoid necrosis, increased syncytial knots, fibrin deposition, and vessel thrombosis.[5] These lead to placental insufficiency and raise the risk of IUGR, preterm birth, and stillbirth. Severity of changes is directly linked to hypertension level, emphasizing the need for early diagnosis and management.[6]

Aim and objectives

Aim

To analyse microscopic changes in the placenta of pregnancy-induced hypertensive (PIH) patients compared to normal pregnancies and correlate these with adverse foetal outcomes.

Objectives

1. Fibrin deposition
2. Fibrinoid necrosis
3. Syncytial knots
4. Cytotrophoblastic cellular proliferation
5. Endothelial proliferation
6. Calcification

MATERIALS AND METHODS

The study was conducted in the Departments of Anatomy and Obstetrics & Gynaecology at B.R.D. Medical College, Gorakhpur, Uttar Pradesh.

- A total of 100 placentas were studied—50 from normal pregnancies and 50 from mothers with pregnancy-induced hypertension (PIH).
- The placentas were collected immediately after delivery from the Obstetrics & Gynaecology Department and further studied in the Anatomy Department.
- Ethical clearance was obtained from Ethical Committee of Baba Raghav Das Medical College, Gorakhpur.

Inclusion Criteria

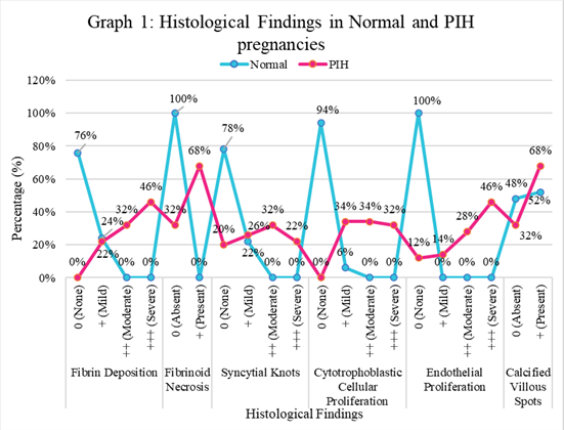
- Placentas were obtained from patients of normal and PIH pregnancies admitted for delivery with gestational age between 37–40 weeks.
- Informed consent was obtained from all participants.

Exclusion Criteria

- Patients with comorbidities such as hypothyroidism, diabetes, or gestational diabetes.
- Patients with positive viral markers.
- Patients with chronic hypertension or hypertension of other causes.

Method: Placentas along with umbilical cords were collected immediately after delivery from both primigravida and multigravida mothers with normal and hypertensive pregnancies. Membranes and cords were trimmed 10 cm from their attachment. Each placenta was washed, blotted dry, then the weight and surface area were measured. The specimens were then fixed in 10% formalin and transported to the Department of Anatomy. After one week of fixation, 1 cm³ tissue samples were taken from maternal and foetal surfaces. The tissues were processed through graded alcohol for dehydration (30% to 97%), cleared using alcohol-xylene mixtures, and blocks were prepared using paraffin wax. Ribbon sections of 7-10µ were obtained using a semi-automatic rotary microtome. The sections were placed on warm water flotation bath at 55°C, and mounted on albumin-coated slides. After drying, the sections were stained with haematoxylin and eosin. Finally, the slides were examined under a light microscope at 100x magnification, and observations were recorded as given in [Table 1].

RESULTS



The above [Graph 1] highlight significant histopathological differences between placentas from normal pregnancies and those affected by pregnancy-induced hypertension (PIH). A marked increase in fibrin deposition was observed in PIH cases, with 100% of placentas showing some degree of deposition, compared to only 24% in normal placentas. In the hypertensive group, severe deposition was noted in 46% of cases, suggesting

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substantial placental ischemia and disrupted maternal-foetal circulation ($p < 0.001$). Fibrinoid necrosis, a hallmark of vascular damage, was completely absent in normal placentas but present in 68% of PIH cases ($p < 0.001$). This indicates widespread endothelial injury and compromised vascular integrity in hypertensive pregnancies. A significant rise in syncytial knots was also evident, with 80% of PIH placentas showing varying degrees, compared to 22% in the normal group ($p < 0.001$). This increase points to placental stress and hypoxia, which are commonly associated with foetal growth restriction and poor neonatal outcomes. In addition, cytotrophoblastic cellular proliferation was observed in 100% of PIH placentas, with moderate to severe proliferation in 66% of cases. This contrasts with only 6% mild proliferation in normal placentas. The finding suggests a compensatory response to maintain placental function under hypoxic conditions ($p < 0.001$). Endothelial proliferation was seen in 88% of PIH placentas, indicating abnormal vascular remodelling, whereas no such change was noted in the control group ($p < 0.001$). Such proliferation may represent an adaptive, yet potentially maladaptive, response to impaired perfusion. Calcified villous spots, indicative of placental aging or degeneration, were significantly more frequent in the PIH group (68%) compared to controls (52%) ($p = 0.042$), further supporting the presence of chronic placental pathology in hypertensive pregnancies.

Table 1: Histopathological Scoring

Fibrin Deposition	
0	Absent
+	Present <20% / lpf
++	Present 20% - 50% / lpf
+++	Present >50% / lpf
Fibrinoid Necrosis	
0	Absent
+	Present
Syncytial Knots	
0	Absent
+	Present <25% / lpf
++	Present >25% / lpf
Cytotrophoblastic Cellular Proliferation	
0	Absent
+	Present <20% / lpf
++	Present 20% - 50% / lpf
+++	Present >50% /lpf
Endothelial Proliferation	
0	Absent
+	Present <20% /lpf
++	Present 20% - 50% /lpf
+++	Present >50% /lpf
Calcification	
0	Absent
+	Present

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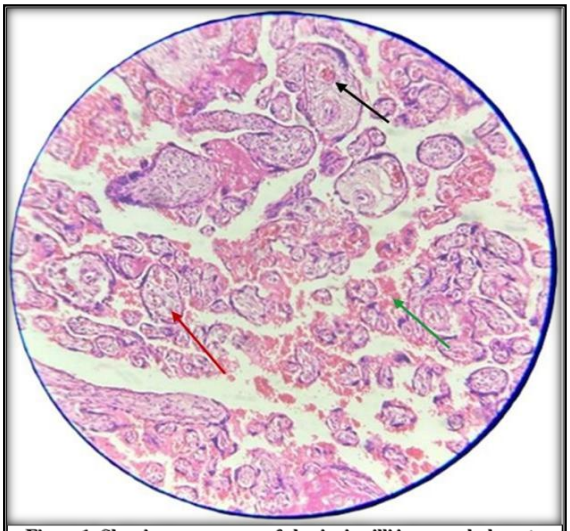

Figure 1- Showing appearance of chorionic villi in normal placenta Red arrow (Chorionic villi having inner core of cytotrophoblast with fetal blood vessels) Black arrow (Fetal blood vessels) Green arrow (Maternal blood between intervillous spaces) at 100x magnification

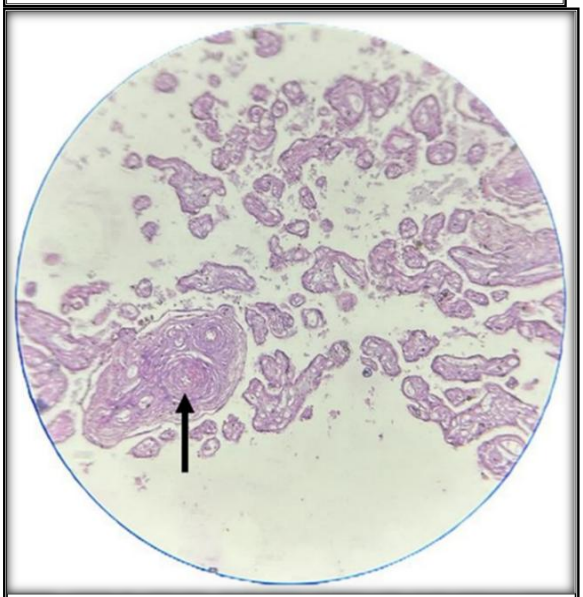

Figure 2- Showing fibrin deposition and hyalinization (+) in chorionic villi of PIH pregnancies at 100x magnification

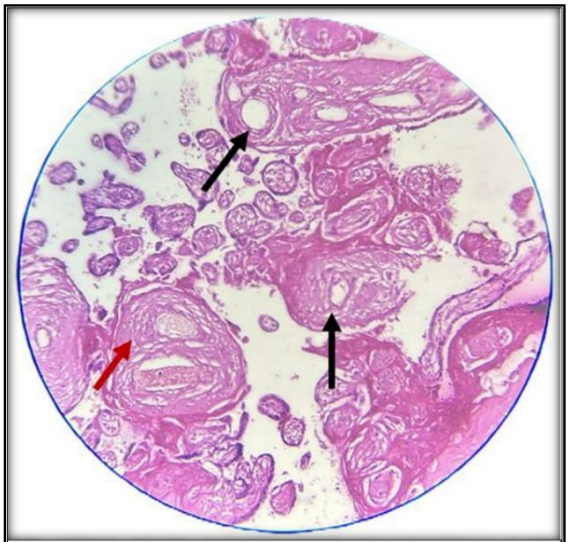

Figure 3- Showing fibrin deposition and hyalinization with Red arrow (+++) and endothelial proliferation with Black arrow (+++) in chorionic villi of PIH pregnancies at 100x magnification

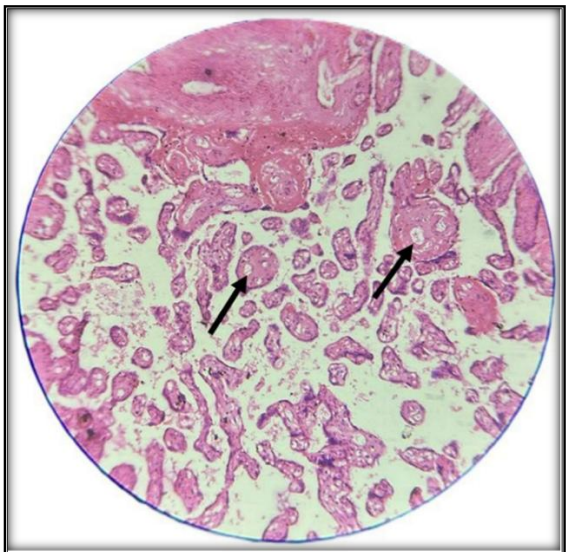

Figure 4- Showing endothelial proliferation (+) in the chorionic villi of PIH pregnancies at 100x magnification

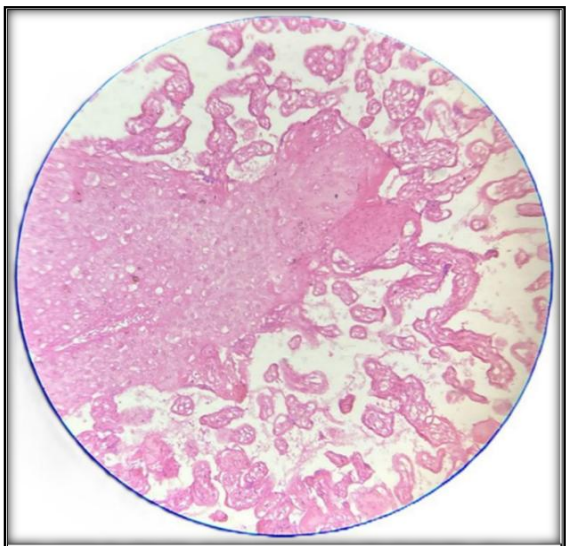

Figure 5- Showing fibrinoid necrosis in the chorionic villi of PIH pregnancies at 100x magnification

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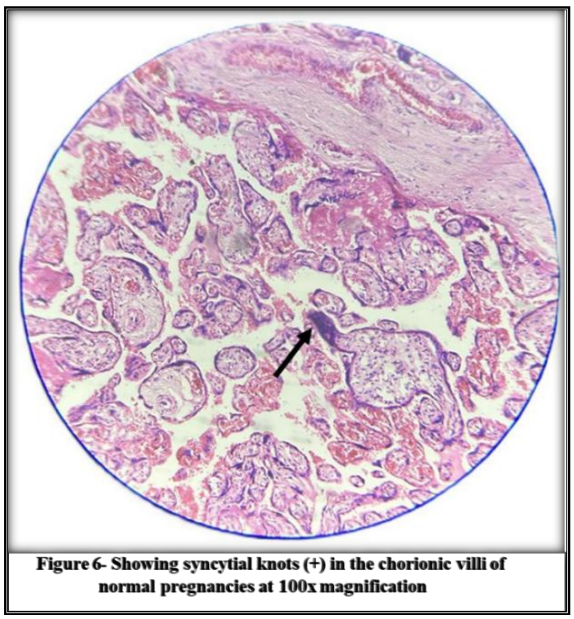

Figure 6- Showing syncytial knots (+) in the chorionic villi of normal pregnancies at 100x magnification

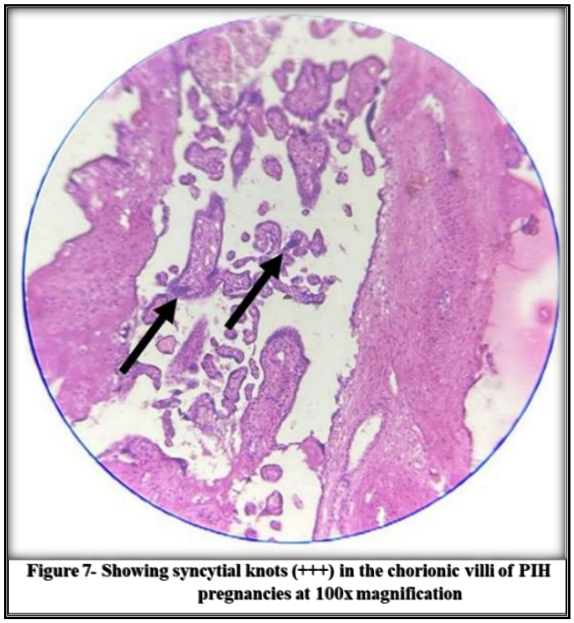

Figure 7- Showing syncytial knots (+++) in the chorionic villi of PIH pregnancies at 100x magnification

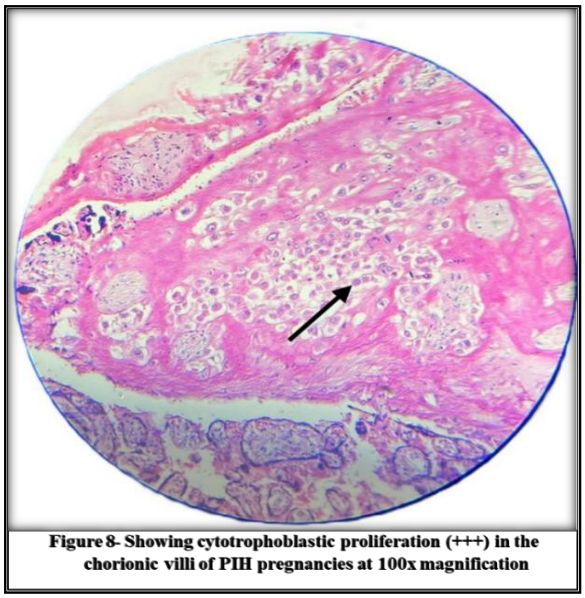

Figure 8- Showing cytotrophoblastic proliferation (+++) in the chorionic villi of PIH pregnancies at 100x magnification

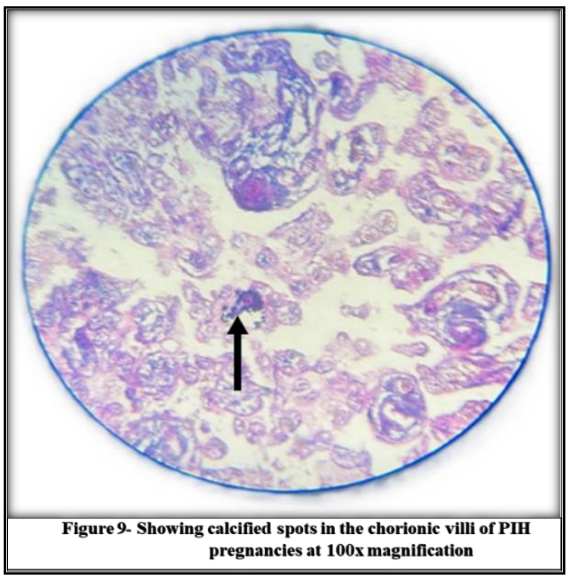

Figure 9- Showing calcified spots in the chorionic villi of PIH pregnancies at 100x magnification

DISCUSSION

The significant histological differences between normal and PIH placentas, with PIH cases showing markedly higher levels of fibrin deposition, fibrinoid necrosis, syncytial knots, cytotrophoblastic and endothelial proliferation, and calcification.

Fibrin deposition, indicative of placental ischemia, was observed in 100% of PIH cases in our study. This aligns with Manjusha et al. (2022), who reported 88%, and exceeds the 36.7% seen in Nimisha Sharma et al. (2023), suggesting more pronounced vascular compromise in our study.[7,8] In contrast, only 24% of normal placentas in our study exhibited mild fibrin deposition, comparable to findings by Siva Sree Ranga et al. (2017), who noted no fibrin in normal placentas.[9]

Fibrinoid necrosis, a marker of endothelial damage, was present in 68% of PIH placentas in our study, similar to the 65% reported by Roberts et al. (1989) and 63.3% by Siva Sree Ranga et al.[9,10] This finding reinforces its association with vascular dysfunction in hypertensive pregnancies. Normal placentas consistently showed absence of necrosis across most studies.

Syncytial knots were found in 80% of PIH placentas in the present study, consistent with findings from Vishram Singh et al. (2021) and Roberts et al. (1989).[10,11] Their increased presence reflects chronic hypoxia and placental stress in PIH, which can adversely affect foetal outcomes.

Cytotrophoblastic proliferation was seen in 100% of PIH placentas in our study, indicating increased cellular activity possibly as a compensatory mechanism in response to hypoxic stress. This finding is comparable to Vishram Singh et al. (2021), who reported 98.68% proliferation in PIH cases, while normal placentas in both studies showed minimal proliferation.[11]

Endothelial proliferation, absent in all normal placentas in our study, was significantly increased in PIH cases (88%), indicating altered placental

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vasculature and adaptive remodeling due to reduced blood flow. This trend closely aligns with Vishram Singh et al., who observed 100% endothelial proliferation in PIH cases and none in controls.[11] Such proliferation reflects endothelial dysfunction and compromised placental perfusion, hallmarks of hypertensive disorders.

Calcification, a marker of placental aging and ischemia, was observed in 68% of PIH placentas in our study, higher than the 52% noted by Roberts et al. (1989) and similar to findings by Siva Sree Ranga et al. (2017).[9,10] Its presence in only 52% of normal placentas further supports its association with pathological placental changes in PIH.

CONCLUSION

The study highlights significant microscopic alterations in placentas from pregnancies complicated by pregnancy-induced hypertension (PIH) compared to normal pregnancies. These histological changes reflect underlying pathophysiological disturbances such as ischemia, vascular dysfunction, and trophoblastic stress, which are central to the adverse outcomes associated with PIH. A consistent and marked increase in fibrin deposition in PIH placentas underscores the role of placental ischemia and impaired maternal-fetal circulation. Similarly, the presence of fibrinoid necrosis in a significant proportion of PIH cases, and its complete absence in normal placentas, strongly indicates endothelial damage and vascular compromise specific to hypertensive disorders. The increased incidence of syncytial knots in PIH placentas further confirms ongoing placental stress, likely a response to chronic hypoxia and reduced perfusion, both of which are associated with intrauterine growth restriction and fetal distress. Additionally, elevated cytotrophoblastic and endothelial proliferation in PIH cases points toward an attempted, yet often insufficient, compensatory response to maintain placental function under hypoxic conditions. This abnormal proliferation may exacerbate placental dysfunction, contributing to poor perinatal outcomes. The significantly higher frequency of calcification in PIH placentas suggests premature placental aging and degenerative changes that further impair exchange capacity. These findings reinforce the critical role of placental pathology in the pathogenesis and complications of PIH. Early detection and monitoring of these histological changes may aid in better risk stratification and timely intervention to improve maternal and fetal outcomes.

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