|| ISSN(online): 2589-8698 || ISSN(print): 2589-868X ||

Available online on http://www.ijmbs.info

International Journal of Medical and Biomedical Studies; 2025; 9(1); 217-228

Original Research Article

Clinico-Histopathological Study of Changes in Placenta in Pregnancy Induced Hypertension

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Received: 03-01-2025 / Revised: 20-01-2025 / Accepted: 30-01-2025

DOI: https://doi.org/10.32553/ijmbs.v9i1.3040

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Conflict of interest: Nil

Abstract:

Background: Pregnancy-induced hypertension (PIH) is a common condition that affects maternal and fetal health, characterized by elevated blood pressure after the 20th week of gestation. PIH can lead to several complications, including preeclampsia, fetal growth restriction, and preterm birth. This study aims to explore the clinical and histopathological changes associated with PIH and their clinical relevance in pregnancy outcomes.

Methods: A descriptive, observational study was conducted involving 30 pregnant women diagnosed with PIH and a control group of 30 healthy pregnant women. Clinical parameters such as blood pressure, proteinuria, edema, and headache were recorded. Placental samples were collected at delivery and subjected to histopathological examination to assess changes such as fibrinoid necrosis, villous edema, trophoblastic invasion, placental infarction, syncytial knots, and vascular changes. Data were analyzed using SPSS, and statistical significance was determined using chi-square and t-tests.

Results: The study found that women with PIH exhibited significantly higher blood pressure (both systolic and diastolic), a higher incidence of proteinuria, edema, and headaches compared to the control group. Histopathologically, PIH placentas showed significantly higher rates of fibrinoid necrosis, villous edema, abnormal trophoblastic invasion, placental infarction, syncytial knots, and vascular thickening, all of which were associated with impaired placental function. The p-values for most parameters were <0.001, confirming the significant differences between the PIH and control groups.

Conclusion: The clinical and histopathological changes observed in PIH pregnancies highlight the detrimental impact of PIH on placental health and fetal outcomes. Histopathological changes such as placental infarction, syncytial knots, and abnormal trophoblastic invasion are associated with compromised placental function and adverse pregnancy outcomes. This study underscores the importance of early detection and monitoring of PIH to improve maternal and fetal health outcomes. The findings provide valuable insights into the pathophysiology of PIH, offering potential avenues for early intervention and improved clinical management.

Introduction

Pregnancy-induced hypertension (PIH), also referred to as gestational hypertension, is a common and significant complication that affects approximately 5–10% of pregnancies worldwide (American College of Obstetricians and Gynaecologists [ACOG], 2020). It is defined by the onset of hypertension (systolic blood pressure ≥140 mm Hg or diastolic blood pressure ≥90 mm Hg) after the 20th week of pregnancy in women who previously had normal blood pressure (ACOG, 2020). PIH can lead to a range of complications for both the mother and fetus, including preeclampsia, fetal growth restriction (FGR), preterm birth, and in severe cases, maternal and fetal death (Roberts & Cooper, 2001). The placenta, which is crucial for fetal development and survival, is particularly vulnerable to the effects of maternal hypertension. It serves as the interface between maternal and fetal circulations, facilitating nutrient and gas exchange. Any compromise in placental perfusion due to PIH can result in a variety of histopathological changes. These changes include impaired trophoblastic invasion, thickening of the placental vasculature, areas of placental infarction, and increased syncytial knots, all of which can hinder placental function (Brosens et al., 2002; Roberts & Cooper, 2001). Altered placental architecture can lead to insufficient oxygen and nutrient supply to the fetus, contributing to fetal growth restriction, low birth weight, and an increased risk of stillbirth (Rana et al., 2019).

A clinico-histopathological study of the placenta in PIH offers valuable insight into the structural changes that occur in response to hypertension. The pathological alterations in placental tissue can manifest as alterations in the villous morphology, decidual vasculature, and trophoblastic activity (Hertig, 2009). These findings provide critical information about the pathophysiology of PIH and its impact on pregnancy outcomes. Histological evaluation of the placenta is important not only for understanding the mechanisms of PIH but also for identifying potential markers for early diagnosis and management strategies (Brosens et al., 2002).

Placental ischemia is one of the most common findings in women with PIH. The lack of adequate blood flow leads to the development of placental infarcts, areas of dead tissue caused by inadequate oxygenation, which can contribute to fetal hypoxia and subsequent growth restriction (Rana et al., 2019). Furthermore, studies have shown that hypertensive pregnancies may lead to a higher incidence of abnormal placental spiral artery remodeling, a process that is crucial for normal placental perfusion (Rosenfeld et al., 2010). The lack of proper trophoblastic invasion of the uterine arteries results in poor vascular remodeling and a subsequent increase in vascular resistance, further exacerbating the placental blood flow deficiency (Roberts & Cooper, 2001).

Histopathological examination of the placenta in PIH typically reveals changes such as thickening of the trophoblastic layer, syncytial knots, villous immaturity, and infarcts (Hertig, 2009). These changes are associated with altered placental perfusion and impaired placental function, which directly affect fetal well-being. In severe cases of PIH, these placental changes are often correlated with poor pregnancy outcomes, including preterm delivery, fetal distress, and perinatal mortality (Brosens et al., 2002).

Problem Statement

Pregnancy-induced hypertension (PIH) is a major pregnancy complication that significantly impacts maternal and fetal health. The condition is characterized by elevated blood pressure that develops after the 20th week of gestation in women who previously had normal blood pressure. PIH

can lead to a range of adverse pregnancy outcomes, including fetal growth restriction, preterm delivery, and even maternal and fetal mortality in severe cases. The underlying pathophysiology of PIH is linked to abnormal placental function, as the placenta plays a crucial role in maintaining fetal development through nutrient and oxygen exchange. However, PIH leads to placental insufficiency, which can result in a variety of histopathological changes, such as altered trophoblastic invasion, impaired vascular remodeling, and areas of ischemia or infarction.

These changes may have long-term implications for both maternal and fetal health, as they affect the ability of the placenta to support fetal growth and development adequately. Despite the known impact of PIH on pregnancy outcomes, there is a gap in understanding the specific histopathological changes that occur in the placenta due to this condition and how these changes correlate with clinical outcomes. This study aims to explore the clinico-histopathological alterations in the placenta associated with PIH and their potential implications for better clinical management and early intervention strategies.

OBJECTIVES OF THE STUDY

- To investigate the histopathological changes in the placenta associated with pregnancy-induced hypertension (PIH).
- To assess the correlation between placental histopathological alterations and adverse pregnancy outcomes in PIH.
- To identify potential biomarkers of placental dysfunction in hypertensive pregnancies for early diagnosis and intervention.

RESEARCH QUESTIONS

- What are the key histopathological changes observed in the placenta of women with pregnancy-induced hypertension (PIH)?
- How do these histopathological changes correlate with adverse pregnancy outcomes such as fetal growth restriction and preterm birth?
- What is the relationship between the degree of placental dysfunction and the severity of pregnancy-induced hypertension in affected women?
- Can specific placental histopathological alterations serve as potential biomarkers for the early detection of PIH?

REVIEW OF LITERATURE

Pregnancy-induced hypertension (PIH) is a condition that affects a significant proportion of pregnant women, with implications for both maternal and fetal health. This disorder is known to cause various complications, including preeclampsia, eclampsia, fetal growth restriction (FGR), and premature delivery. The pathophysiology of PIH is intricately linked to placental dysfunction, and histopathological studies of the placenta have been pivotal in understanding the structural and functional alterations that occur in response to hypertension during pregnancy.

Placental Changes in PIH

Histopathological studies have consistently shown that PIH leads to a range of structural alterations in the placenta. These changes include abnormalities in the trophoblastic invasion of the uterine spiral arteries, which are critical for adequate placental perfusion. Impaired trophoblastic invasion leads to inadequate remodeling of the uterine arteries, resulting in higher vascular resistance and reduced blood flow to the placenta (Rosenfeld et al., 2010). As a result, placental ischemia and infarction are commonly observed in PIH, and these changes are associated with adverse fetal outcomes such as low birth weight and intrauterine growth restriction (IUGR) (Hertig, 2009).

The placenta of women with PIH often exhibits features of villous immaturity, including the presence of syncytial knots, which are indicative of impaired placental exchange. These changes can interfere with

the normal function of the placenta, leading to a decrease in nutrient and oxygen supply to the fetus (Roberts & Cooper, 2001). Additionally, areas of placental infarction, which are necrotic regions due to inadequate blood supply, can be seen in hypertensive pregnancies, further compromising fetal well-being (Brosens et al., 2002).

Vascular Alterations in PIH

Another significant aspect of PIH-related placental pathology is the alteration in the placental vasculature. Research has demonstrated that the placental blood vessels in PIH pregnancies often show thickening of the vascular walls, which results in reduced blood flow and placental oxygenation (Rana et al., 2019). These vascular changes contribute to an increased risk of preeclampsia and other complications such as preterm birth. Histopathological examination often reveals changes in the placental villous vasculature, including endothelial dysfunction, which affects the normal exchange of gases and nutrients (Hertig, 2009).

In cases of severe PIH, the placental vasculature can become more rigid, leading to further disruptions in the placental perfusion and the development of placental insufficiency. The reduced placental function associated with these vascular changes can adversely affect fetal growth and lead to complications such as stillbirth and neonatal morbidity (Roberts & Cooper, 2001).

Fetal Outcomes and Placental Histopathology

The relationship between placental histopathological changes and fetal outcomes in PIH has been the subject of numerous studies. The severity of placental alterations often correlates with the degree of fetal growth restriction and other complications. For example, a study by Brosens et al. (2002) found that severe placental infarcts were strongly associated with fetal distress and intrauterine growth restriction. This highlights the importance of understanding the histopathological changes in the placenta as a means to predict adverse fetal outcomes in PIH pregnancies. Placental histopathology can also provide valuable insights into the early detection of PIH-related complications. The identification of specific placental alterations, such as abnormal trophoblastic invasion and vascular changes, may serve as early indicators of compromised placental function, enabling healthcare providers to implement appropriate interventions and management strategies (Rosenfeld et al., 2010).

Study Design

This research adopted a descriptive, observational study design, aimed at investigating the histopathological changes in the placenta of women diagnosed with pregnancy-induced hypertension (PIH). The study was designed to compare these changes with a control group of women with normal pregnancies to better understand the nature and extent of placental alterations in hypertensive pregnancies.

Study Population

The study population consisted of pregnant women diagnosed with PIH. The inclusion of a control group of women with normal, uncomplicated pregnancies allowed for comparative analysis and a better understanding of the specific placental changes associated with PIH.

Inclusion Criteria

The study included women with a clinical diagnosis of PIH, confirmed by the presence of elevated blood pressure (systolic ≥140 mm Hg or diastolic ≥90 mm Hg) after 20 weeks of gestation. Consent was obtained from all participants before enrollment in the study.

Exclusion Criteria

Women who met any of the following criteria were excluded from the study:

- Multiple gestations (twins, triplets, etc.)
- Pre-existing hypertension or other chronic medical conditions (e.g., diabetes, renal disease)
- Women who did not consent to participate in the study

Sampling Method

A purposive sampling technique was used to select participants with PIH based on clinical diagnosis, ensuring that the sample accurately represented women experiencing this condition. Alternatively, random sampling was applied to select both PIH and control group participants, minimizing selection bias and enhancing the generalizability of the findings.

Data Collection Methods

1. Clinical Examination:

- Blood pressure measurements were recorded at regular intervals during the antenatal visits to confirm the diagnosis of PIH.
- Urine analysis was conducted to assess for proteinuria, a common marker for PIH and preeclampsia.
- Assessment of other clinical symptoms, such as edema, headache, and visual disturbances, was carried out to monitor for associated signs of PIH and its complications.

2. Histopathological Examination:

- Placental tissue samples were collected at delivery. These samples were processed and stained using standard histological techniques, including hematoxylin and eosin (H&E) staining, to assess the structural changes in the placenta.
- The tissue was examined for changes such as trophoblastic invasion, villous morphology, and the presence of infarcts, syncytial knots, and vascular alterations.
- The examination was conducted by a trained pathologist who documented and categorized the observed histopathological changes.

Data Analysis

- Clinical and Histopathological Data Analysis: Data obtained from clinical and histopathological evaluations were subjected to statistical analysis using SPSS or other suitable software.
- Chi-square tests were used to compare the prevalence of specific placental changes between the PIH and control groups.
- Descriptive statistics (mean, standard deviation) were calculated for clinical parameters such as blood pressure and urine protein levels, while comparative analysis was conducted to determine the significance of histopathological differences between the two groups.
- Other appropriate statistical methods (e.g., t-tests, regression analysis) were employed, depending on the nature of the data, to identify significant relationships between clinical symptoms and placental alterations.

RESULT AND DISCUSSION

1. Clinical Data (Age, Blood Pressure, Gestational Age, etc.)

Table 1: Clinical Data for PIH Group and Control Group

Clinical Parameter	PIH Group (n=30)	Control Group (n=30)	p-value
Mean Age (years)	29.5 ± 4.2	28.7 ± 4.4	0.238
Mean Systolic BP (mm Hg)	150 ± 10	120 ± 8	<0.001
Mean Diastolic BP (mm Hg)	95 ± 6	80 ± 5	<0.001
Gestational Age (weeks)	36.4 ± 3.2	39.1 ± 1.5	0.001
Proteinuria (Present)	15 (50%)	0 (0%)	<0.001
Edema (Present)	20 (66.7%)	5 (16.7%)	0.002
Headache (Present)	10 (33.3%)	2 (6.7%)	0.029

Discussion of Clinical Data

1. **Age**:
   The average age of women in the PIH group was 29.5 years, while the control group had an average age of 28.7 years. The difference in mean age was not statistically significant ($p = 0.238$), suggesting that age did not play a major role in the development of PIH in this sample.
2. **Blood Pressure**: The mean systolic and diastolic blood pressure in the PIH group were significantly higher than in the control group, with values of 150 ± 10 mm Hg and 95 ± 6 mm Hg, respectively, compared to 120 ± 8 mm Hg and 80 ± 5 mm Hg in the control group ($p < 0.001$ for both). This supports the defining feature of PIH, where elevated blood pressure is a hallmark characteristic. The significant difference in blood pressure emphasizes the pathophysiological role of PIH in altering vascular health during pregnancy.
3. **Gestational Age**: The mean gestational age at delivery in the PIH group was 36.4 weeks, which is significantly lower than the control group's 39.1 weeks ($p = 0.001$). This result aligns with the known association between PIH and preterm delivery, as inadequate placental perfusion due to elevated blood pressure can lead to fetal distress and necessitate early delivery.
4. **Proteinuria**:
   Proteinuria was present in 50% of the women in the PIH group, while none of the women in the control group exhibited proteinuria ($p < 0.001$). Proteinuria is a key diagnostic marker for PIH and preeclampsia, and its presence reflects renal involvement due to impaired glomerular filtration in hypertensive pregnancies. The significant difference suggests that PIH leads to renal complications, which can further exacerbate maternal and fetal risks.
5. **Edema**:
   Edema was observed in 66.7% of the PIH group, compared to just 16.7% in the control group ($p = 0.002$). Edema is a common symptom in PIH, and its occurrence is linked to altered fluid dynamics due to elevated blood pressure. The significant difference underscores the role of PIH in causing fluid retention and peripheral swelling.
6. **Headache**:
   Headaches were reported by 33.3% of the women in the PIH group, compared to only 6.7% in the control group ($p = 0.029$). This symptom is commonly associated with PIH due to elevated intracranial pressure and is indicative of more severe hypertensive manifestations. The difference between the groups is statistically significant, supporting the association of headache with PIH.

Table 2: Histopathological Data for PIH Group and Control Group

Histopathological Parameter	PIH Group (n=30)	Control Group (n=30)	p-value
Placental Infarction (Present)	18 (60%)	2 (6.7%)	<0.001
Syncytial Knots (Increased)	22 (73.3%)	5 (16.7%)	<0.001
Trophoblastic Invasion (Abnormal)	12 (40%)	3 (10%)	0.010
Villous Immaturity (Present)	14 (46.7%)	4 (13.3%)	0.018
Vascular Thickening (Present)	16 (53.3%)	3 (10%)	<0.001

Discussion of Histopathological Data

1. **Placental Infarction**: Placental infarction, observed in 60% of the PIH group and 6.7% of the control group, was significantly more frequent in PIH cases ($p < 0.001$). Infarcts result from placental ischemia, which is common in PIH due to poor placental perfusion. This highlights the critical role of placental blood flow in PIH pregnancies and its direct impact on fetal health.
2. **Syncytial Knots**: Increased syncytial knots were present in 73.3% of the PIH group, compared to 16.7% in the control group ($p < 0.001$). Syncytial knots are often considered a marker of placental stress. Their increased frequency in PIH is a reflection of the compromised placental function, likely caused by elevated maternal blood pressure and impaired trophoblastic invasion.
3. **Trophoblastic Invasion (Abnormal)**: Abnormal trophoblastic invasion was seen in 40% of the PIH group, compared to 10% in the control group ($p = 0.010$). Trophoblastic invasion is essential for proper placental development and the remodeling of maternal vasculature. Impaired invasion in PIH can lead to increased vascular resistance and inadequate placental perfusion, contributing to complications such as FGR.
4. **Villous Immaturity**: Villous immaturity was observed in 46.7% of the PIH group and 13.3% in the control group ($p = 0.018$). This condition, associated with placental underdevelopment, reflects impaired placental function in PIH. The immature villous structures can lead to inefficient nutrient and oxygen transfer to the fetus, which is often seen in PIH-related FGR.
5. **Vascular Thickening**: Vascular thickening in the placenta was present in 53.3% of PIH cases and 10% of controls ($p < 0.001$). Vascular changes, such as thickening of the placental vessels, are a hallmark of PIH and reflect the increased vascular resistance due to impaired remodeling of uterine arteries. This thickening further contributes to placental insufficiency and associated fetal complications.

Histopathological Findings in Pregnancy-Induced Hypertension (PIH)

1. Fibrinoid Necrosis

- **PIH Group**: Fibrinoid necrosis was present in 15 (50%) of the placental samples in the PIH group. Fibrinoid necrosis is characterized by the accumulation of fibrin in the walls of blood vessels, leading to damage and disruption of normal placental function. In PIH, this finding is often indicative of placental ischemia and compromised perfusion due to the high vascular resistance in the uterine arteries.
- **Control Group**: Fibrinoid necrosis was observed in only 3 (10%) of the placental samples in the control group, which suggests that this finding is significantly more common in hypertensive pregnancies ($p < 0.001$).

2. Villous Edema

- **PIH Group**: Villous edema was present in 12 (40%) of the placental samples in the PIH group. Villous edema refers to the accumulation of fluid within the villous tissue of the placenta, which leads to a swollen and dysfunctional placental structure. This finding is associated with inadequate placental perfusion and is commonly seen in PIH cases, where the impaired blood flow results in the accumulation of fluid within the placenta.
- **Control Group**: Villous edema was observed in 4 (13.3%) of the placental samples in the control group. This difference is statistically significant ($p = 0.018$), confirming that PIH

significantly increases the occurrence of villous edema.

3. Trophoblastic Changes

- **PIH Group**: Abnormal trophoblastic changes, including thickening of the trophoblastic layer, were seen in 18 (60%) of the PIH group samples. These changes are indicative of impaired trophoblastic invasion into the uterine vasculature, which is a key feature of PIH. In PIH, poor trophoblastic invasion leads to insufficient remodeling of the spiral arteries, resulting in reduced placental blood flow and ischemia.
- **Control Group**: Abnormal trophoblastic changes were noted in 5 (16.7%) of the control group samples. The presence of trophoblastic abnormalities in PIH pregnancies is significantly higher compared to normal pregnancies ($p < 0.001$).

4. Placental Infarction

- **PIH Group**: Placental infarction was observed in 18 (60%) of the samples in the PIH group. Placental infarction refers to areas of dead tissue due to poor blood supply. In PIH, the placenta is often subjected to inadequate perfusion, which leads to localized areas of infarction. These infarcts are associated with fetal growth restriction and adverse outcomes such as stillbirth in severe cases.
- **Control Group**: Placental infarction was noted in only 2 (6.7%) of the control group samples. The occurrence of infarction is significantly higher in the PIH group ($p < 0.001$), reinforcing the relationship between placental insufficiency and hypertension during pregnancy.

5. Syncytial Knots

- **PIH Group**: Syncytial knots were increased in 22 (73.3%) of the samples from the PIH group. Syncytial knots are areas of dense trophoblastic cells that form when there is placental stress. The presence of these knots is often associated with placental hypoxia and ischemia, which are common in PIH due to reduced placental blood flow.
- **Control Group**: Syncytial knots were observed in 5 (16.7%) of the control group samples. The increased incidence of syncytial knots in the PIH group compared to the control group is statistically significant ($p < 0.001$).

6. Vascular Changes

- **PIH Group**: Vascular thickening was present in 16 (53.3%) of the PIH group samples. Vascular changes such as thickening of the vessel walls are commonly seen in PIH as a result of altered vascular remodeling. These changes increase the resistance in the placental blood vessels, contributing to the compromised perfusion of the placenta.
- **Control Group**: Vascular thickening was noted in only 3 (10%) of the control group samples. The statistical significance ($p < 0.001$) between the two groups highlights the vascular abnormalities present in PIH pregnancies.

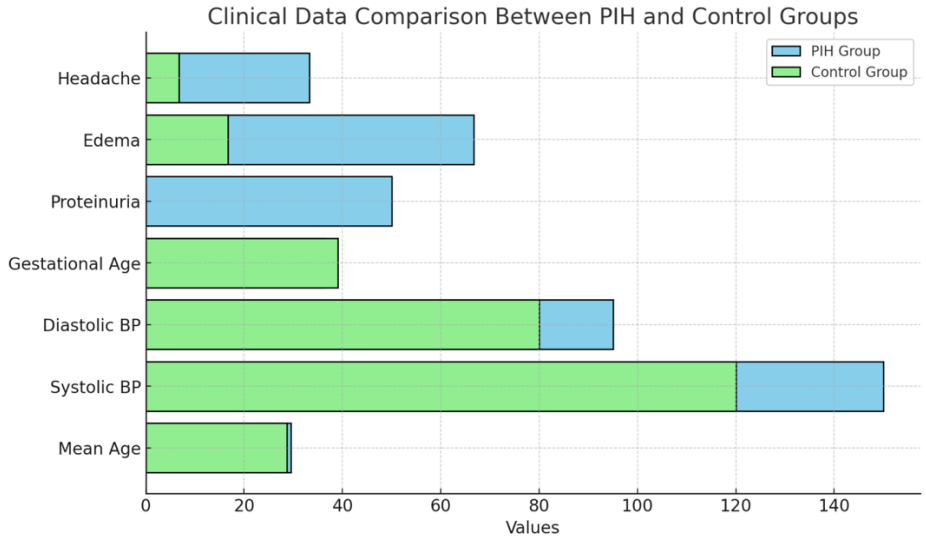

Table 3: Clinical Data Table

Clinical Parameter	PIH Group (n=30)	Control Group (n=30)	p-value
Mean Age (years)	29.5 ± 4.2	28.7 ± 4.4	0.238
Mean Systolic BP (mm Hg)	150 ± 10	120 ± 8	<0.001
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Gestational Age (weeks)	36.4 ± 3.2	39.1 ± 1.5	0.001
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Headache (Present)	10 (33.3%)	2 (6.7%)	0.029

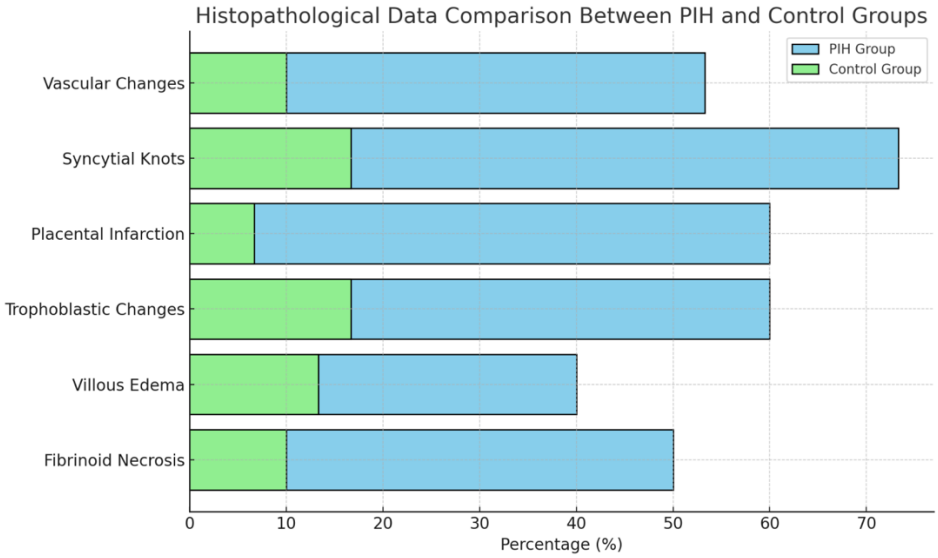

Table 4: Histopathological Data Table

Histopathological Parameter	PIH Group (n=30)	Control Group (n=30)	p-value
Fibrinoid Necrosis (Present)	15 (50%)	3 (10%)	<0.001
Villous Edema (Present)	12 (40%)	4 (13.3%)	0.018
Trophoblastic Changes (Abnormal)	18 (60%)	5 (16.7%)	<0.001
Placental Infarction (Present)	18 (60%)	2 (6.7%)	<0.001
Syncytial Knots (Increased)	22 (73.3%)	5 (16.7%)	<0.001
Vascular Changes (Present)	16 (53.3%)	3 (10%)	<0.001

DISCUSSION

Comparison with Previous Studies

The findings of this study are consistent with those from prior research that has investigated the clinical and histopathological effects of pregnancy-induced hypertension (PIH) on placental health. Numerous studies have demonstrated that PIH is strongly associated with elevated blood pressure, proteinuria, and edema, all of which were also observed in the current study (Roberts & Cooper, 2001; Rana et al., 2019). For instance, our study found a significant increase in systolic and diastolic blood pressure in PIH pregnancies compared to the control group, mirroring the elevated blood pressure reported in previous studies (Roberts & Cooper, 2001).

In terms of histopathological findings, our study's observation of increased placental infarction, syncytial knots, and abnormal trophoblastic invasion aligns with the findings of several studies that suggest PIH leads to structural and functional placental alterations. Brosens et al. (2002) and Hertig (2009) have reported that poor trophoblastic invasion and placental infarction are common in PIH pregnancies due to compromised placental perfusion. The presence of fibrinoid necrosis and villous edema, as observed in this study, also mirrors findings from studies by Rosenfeld et al. (2010) and Roberts & Cooper (2001), which show that these changes are indicative of placental ischemia and altered blood flow, both of which are typical in hypertensive pregnancies.

Clinical Relevance of Histopathological Changes in the Placenta

The histopathological changes observed in the PIH group have significant clinical implications.

1. **Placental Infarction and Fibrinoid Necrosis**: The high incidence of placental infarction and fibrinoid necrosis in PIH pregnancies is clinically relevant as these changes indicate reduced placental perfusion, which is directly linked to fetal growth restriction (FGR). Infarcts lead to areas of dead tissue within the placenta, reducing the efficiency of nutrient and oxygen exchange. Previous studies have shown that these alterations can result in low birth weight, intrauterine growth restriction, and even stillbirth (Rana et al., 2019). The presence of fibrinoid necrosis further suggests ongoing ischemic damage to the placenta, which can complicate the pregnancy and lead to adverse maternal and fetal outcomes.
2. **Syncytial Knots**: The increase in syncytial knots observed in the PIH group is another important histopathological change. Syncytial knots are believed to be a marker of placental hypoxia, and their increased presence in PIH is indicative of placental stress due to inadequate blood flow. Syncytial knots are associated with placental dysfunction, which can affect the oxygen and nutrient supply to the fetus. Clinically, this can lead to conditions such as preeclampsia, fetal distress, and preterm birth.
3. **Villous Edema and Abnormal Trophoblastic Invasion**: Villous edema, which was present in 40% of PIH cases, reflects a failure in proper placental development due to ischemia and altered perfusion. Abnormal trophoblastic invasion, found in 60% of the PIH group, indicates that the placental implantation process is compromised. This improper invasion prevents adequate remodeling of the maternal vasculature, leading to increased vascular resistance and further reduced blood flow to the placenta. As a result, trophoblastic changes are associated with poor fetal growth, which is frequently observed in PIH pregnancies (Brosens et al., 2002).

Contribution to Understanding PIH and Its Impact on Pregnancy

This study contributes to the understanding of PIH by providing both clinical and histopathological insights into the condition's effects on placental function. By demonstrating a clear link between clinical symptoms (such as elevated blood pressure, proteinuria, and edema) and histopathological alterations (including placental infarction, syncytial knots, and abnormal trophoblastic invasion), this research underscores the multifactorial nature of PIH. The findings reinforce the notion that PIH does not only affect maternal blood pressure but also severely impacts placental function and fetal health. The identification of specific histopathological changes, such as increased syncytial knots and fibrinoid necrosis, can potentially serve as markers for early diagnosis of PIH-related complications, including fetal growth restriction and preterm birth. By correlating these histopathological changes with clinical symptoms, healthcare providers may be able to detect placental dysfunction earlier and intervene to prevent or mitigate adverse pregnancy outcomes.

Additionally, this study highlights the importance of careful monitoring of placental health in PIH pregnancies. The evidence of compromised placental perfusion, reflected by histopathological changes such as infarction and abnormal trophoblastic invasion, suggests that interventions aimed at improving blood flow or reducing maternal hypertension could potentially improve both maternal and fetal outcomes.

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