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

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

International Journal of Medical and Biomedical Studies; 2025; 9(2); 92-103

Original Research Article

Correlation of Placental Morphological Changes with Fetal Outcome in Pregnancy Induced Hypertension

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Received: 19-01-2025 / Revised: 04-02-2025 / Accepted: 11-03-2025

DOI: https://doi.org/10.32553/ijmbs.v9i2.3039

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Conflict of interest: No conflict of interest

Abstract:

Background: Pregnancy-induced hypertension (PIH) is a common pregnancy complication associated with significant maternal and fetal risks. The placental abnormalities caused by PIH, such as infarctions, villous edema, and fibrinoid necrosis, are believed to affect fetal development and lead to adverse outcomes like low birth weight, intrauterine growth restriction (IUGR), and neonatal complications. This study aimed to explore the correlation between placental morphological changes and fetal outcomes in pregnancies complicated by PIH.

Methods: A descriptive observational study was conducted, including 30 women diagnosed with PIH and 30 women with normal pregnancies as the control group. Placental morphological changes, such as infarctions, villous edema, and fibrinoid necrosis, were examined post-delivery. Fetal outcomes, including birth weight, Apgar scores, and neonatal complications, were recorded. Statistical analyses, including Pearson's correlation and multiple regression analysis, were performed to assess the relationships between placental changes and fetal outcomes.

Results: The study found significant correlations between placental infarctions and reduced birth weight ($r = -0.72$, $p < 0.01$), villous edema and lower Apgar scores ($r = -0.65$, $p < 0.05$), and fibrinoid necrosis with neonatal complications ($r = 0.80$, $p < 0.01$). Placental weight was positively correlated with fetal birth weight ($r = 0.65$, $p < 0.05$). Regression analyses indicated that placental infarctions and placental weight were significant predictors of fetal birth weight, while fibrinoid necrosis predicted neonatal complications.

Conclusion: This study highlights the significant role of placental morphological changes in predicting fetal outcomes in PIH pregnancies. Placental infarctions, villous edema, and fibrinoid necrosis were identified as key markers of poor fetal growth, low Apgar scores, and neonatal complications. Early detection of these placental abnormalities through regular monitoring could help in better management of PIH pregnancies and improve maternal and fetal outcomes.

Keywords: Pregnancy-induced hypertension, placental infarctions, villous edema, fibrinoid necrosis, fetal outcomes, birth weight, Apgar scores, neonatal complications.

Introduction

Pregnancy-induced hypertension (PIH) is a condition that manifests as high blood pressure after 20 weeks of pregnancy, affecting a significant number of pregnancies worldwide. This condition is a major cause of maternal morbidity and mortality, contributing to complications such as preeclampsia, placental abruption, and increased risk of cardiovascular diseases later in life. PIH is associated with poor fetal outcomes, including intrauterine growth restriction (IUGR), preterm birth, and fetal distress, making it a critical area of investigation in obstetrics and maternal-fetal medicine (Sibai et al., 2015). The placenta, the organ responsible for nourishing the fetus, is significantly impacted in cases of PIH. Placental insufficiency, resulting from impaired placental blood flow and abnormal trophoblastic invasion, can lead to a variety of morphological changes in the placenta, such as increased infarctions, fibrosis, and villous abnormalities. These changes can have direct consequences on fetal health, as they are often linked to impaired nutrient and oxygen transfer from the mother to the fetus (Challier et al., 2008).

Understanding the correlation between placental morphological changes and fetal outcomes is vital, as it provides insights into the underlying pathophysiology of PIH and allows for better management strategies. In PIH, placental abnormalities can manifest in various forms, including smaller placental size, increased rate of infarctions, abnormal villous architecture, and fibrinoid necrosis (Roberts & Cooper, 2001).

These changes are often indicative of a placental failure to adequately perfuse the fetus, leading to insufficient fetal development and compromised health (Villar et al., 2006). Studies have consistently shown that PIH leads to vascular and trophoblastic abnormalities that affect the placental structure. For example, the trophoblast cells, which normally invade the uterine wall and promote the formation of maternal blood vessels, fail to invade properly in PIH, leading to an increased risk of placental hypoperfusion. This lack of proper perfusion is believed to result in increased oxidative stress, which damages placental tissues and disrupts fetal development (Vaughan et al., 2017).

These placental changes in PIH are associated with fetal complications, including intrauterine growth restriction (IUGR), which occurs when the fetus is unable to grow at a normal rate due to inadequate nutrient and oxygen supply. IUGR is one of the most serious outcomes of PIH, as it increases the risk of preterm birth, low birth weight, and perinatal mortality. In addition to growth restriction, PIH has been linked to an increased incidence of fetal hypoxia, which can lead to fetal distress and even stillbirth in severe cases (Sibai et al., 2015).

The relationship between placental morphology and fetal outcomes in PIH is complex and multifactorial. Morphological changes in the placenta are not always directly indicative of the severity of fetal compromise, as some fetuses may experience poor outcomes despite relatively mild placental abnormalities. However, the presence of certain placental features, such as significant infarctions or villous necrosis, can often serve as indicators of poor fetal prognosis (Villar et al., 2006). Furthermore, studies have suggested that a combination of maternal risk factors, such as obesity, advanced maternal age, and pre-existing hypertension, can exacerbate the effects of PIH on both the placenta and the fetus (Challier et al., 2008).

This study aims to examine the correlation between placental morphological changes and fetal outcomes in pregnancies complicated by PIH. By analyzing the relationship between the extent of placental

alterations and the severity of fetal compromise, this research will contribute to a better understanding of the pathophysiology of PIH and may offer valuable insights for improving prenatal care and pregnancy management.

PROBLEM OF THE STATEMENT

Pregnancy-induced hypertension (PIH) is a leading cause of maternal and fetal complications worldwide, affecting a significant proportion of pregnancies. PIH is associated with several adverse outcomes, including preeclampsia, intrauterine growth restriction (IUGR), preterm birth, and even stillbirth. These complications often arise due to placental insufficiency, which is a direct consequence of impaired placental function and morphology in PIH cases. The placenta, being crucial for nutrient and oxygen exchange, undergoes various morphological changes under the stress of hypertension.

These alterations include reduced trophoblastic invasion, placental infarctions, villous abnormalities, and decreased placental size, all of which can impede the proper development of the fetus. Despite the well-established association between PIH and poor fetal outcomes, there is limited research focusing on the direct correlation between specific placental morphological changes and fetal health. The current understanding of this relationship remains incomplete, with some studies suggesting that placental changes do not always correlate directly with the severity of fetal compromise. Additionally, there is a lack of comprehensive studies that compare the spectrum of placental abnormalities in PIH pregnancies with the range of adverse fetal outcomes observed, such as IUGR, low birth weight, and fetal distress.

This gap in the literature creates a pressing need to investigate the correlation between placental morphological changes and fetal outcomes in PIH, aiming to improve the prediction and management of PIH-related pregnancies. Identifying which placental features are most indicative of poor fetal outcomes could enhance prenatal monitoring, enable early interventions, and ultimately reduce the risks for both mothers and their babies. Therefore, this study aims to explore how placental morphological alterations in PIH correlate with fetal health and contribute to adverse pregnancy outcomes, offering a more comprehensive understanding of the pathophysiology underlying PIH and its impact on maternal and fetal well-being.

OBJECTIVES OF THE STUDY

- To examine the correlation between placental morphological changes and fetal outcomes in pregnancies complicated by pregnancy-induced hypertension (PIH).
- To identify specific placental features associated with adverse fetal outcomes such as intrauterine growth restriction (IUGR) and preterm birth in PIH.
- To evaluate the impact of placental abnormalities on fetal health, including fetal distress and low birth weight, in PIH-affected pregnancies.

RESEARCH QUESTIONS

- What is the relationship between placental morphological changes and fetal outcomes in pregnancies complicated by pregnancy-induced hypertension (PIH)?
- Which specific placental features are most strongly associated with adverse fetal outcomes such as intrauterine growth restriction (IUGR) and preterm birth in PIH?
- How do placental abnormalities in PIH-affected pregnancies influence fetal health, including fetal distress and low birth weight?
- Can placental morphological changes serve as reliable predictors of fetal complications in pregnancies complicated by PIH?

REVIEW OF LITERATURE

Pregnancy-induced hypertension (PIH) is one of the most common pregnancy complications, often leading to significant maternal and fetal morbidity. The placenta, which is essential for nutrient and oxygen exchange between mother and fetus, undergoes various changes in PIH that may have critical implications for fetal development and outcome. Several studies have investigated the relationship between placental abnormalities and fetal outcomes in PIH, suggesting that morphological changes in the placenta are associated with increased risk for adverse pregnancy outcomes such as intrauterine growth restriction (IUGR), preterm birth, and fetal distress.

Placental Changes in PIH

In PIH, the placenta frequently exhibits abnormal morphology, including reduced size, infarctions, villous atrophy, and fibrinoid necrosis. These changes can be attributed to impaired trophoblastic invasion and abnormal vascular remodeling (Sibai et al., 2015). Trophoblastic cells, which normally invade the uterine wall to ensure adequate blood flow, fail to infiltrate sufficiently in PIH, leading to a reduced supply of oxygen and nutrients to the fetus (Roberts & Cooper, 2001). This impaired placental perfusion often results in pathological changes that can affect fetal development, contributing to conditions such as IUGR, low birth weight, and preterm delivery (Challier et al., 2008).

Impact of Placental Morphology on Fetal Outcome

The morphology of the placenta plays a crucial role in determining fetal outcomes in PIH. Studies have shown that certain placental abnormalities, such as increased placental infarctions and villous necrosis, are closely linked to poor fetal outcomes. For instance, placental infarctions have been associated with fetal growth restriction and other complications, as they signify a disruption in the placenta's ability to provide adequate blood flow and nutrition to the fetus (Villar et al., 2006). Additionally, decreased placental weight, often observed in PIH pregnancies, has been shown to correlate with increased risks of adverse neonatal outcomes (Vaughan et al., 2017).

Fetal Growth Restriction and Placental Abnormalities

Fetal growth restriction (IUGR) is one of the most concerning outcomes in PIH, often associated with significant placental changes. The impaired trophoblastic invasion that occurs in PIH leads to reduced placental perfusion, which in turn results in a lower supply of oxygen and nutrients to the fetus. This can cause IUGR, a condition in which the fetus is unable to grow to its full potential, leading to low birth weight and increased risk of neonatal complications such as respiratory distress and stillbirth (Sibai et al., 2015). Several studies have demonstrated that placental abnormalities such as increased infarctions, reduced villous architecture, and decreased vascularization correlate with higher rates of IUGR in PIH pregnancies (Challier et al., 2008; Roberts & Cooper, 2001).

Other Adverse Pregnancy Outcomes

Besides fetal growth restriction, PIH is also linked to other serious fetal complications, including preterm birth and fetal distress. Preterm birth, which occurs in approximately 10-15% of pregnancies complicated by PIH, is often associated with placental insufficiency and reduced placental function. The compromised nutrient and oxygen exchange in PIH pregnancies increases the risk of premature rupture of membranes and early labor, further exacerbating the risks to both the mother and the fetus (Villar et al., 2006). Furthermore, studies have shown that abnormal placental morphology in PIH pregnancies is strongly associated with fetal hypoxia, a condition in which the fetus is deprived of adequate oxygen, leading to fetal distress and an increased need for

neonatal intensive care (Vaughan et al., 2017).

Predictive Value of Placental Morphological Changes

Identifying the specific placental changes that predict adverse fetal outcomes can greatly improve prenatal care and intervention strategies. Some studies suggest that placental features, such as the presence of infarctions, reduced villous surface area, and altered vascular patterns, can serve as markers for the severity of fetal compromise in PIH pregnancies (Sibai et al., 2015). Additionally, advancements in imaging techniques, such as Doppler ultrasonography, have allowed for better evaluation of placental blood flow and can assist in predicting fetal outcomes based on placental health (Villar et al., 2006).

RESEARCH METHODOLOGY

Study Design

This study adopted a descriptive observational study design to explore the correlation between placental morphological changes and fetal outcomes in pregnancies complicated by pregnancy-induced hypertension (PIH). The study aimed to identify specific placental alterations and how they related to adverse fetal outcomes, such as intrauterine growth restriction (IUGR), low birth weight, and neonatal complications.

POPULATION AND SAMPLE

Inclusion Criteria:

- Pregnant women diagnosed with PIH based on elevated blood pressure (systolic ≥140 mm Hg or diastolic ≥90 mm Hg) after 20 weeks of gestation.
- Women with a single, uncomplicated pregnancy (excluding multiple gestations).
- Women who provided informed consent to participate in the study.

Exclusion Criteria:

- Women with pre-existing hypertension, diabetes, or other chronic medical conditions (e.g., renal disease).
- Pregnancies complicated by other conditions like preeclampsia, eclampsia, or gestational diabetes.
- Women who were unable to provide informed consent.

Sample Size: The sample consisted of 30 women diagnosed with PIH, and a control group of 30 women with normal pregnancies was included to compare the placental changes and fetal outcomes. The sample size was chosen to provide a significant statistical analysis while being feasible within the scope of the study.

DATA COLLECTION

Clinical Data:

- **Blood Pressure:** Regular blood pressure readings were taken throughout pregnancy to monitor the progression of PIH.
- **Gestational Age:** Determined using ultrasound measurements and the last menstrual period, ensuring an accurate assessment of fetal development and outcome.

Placental Data:

- **Morphological Examination:** The placenta was collected at delivery and subjected to a detailed histopathological examination to identify morphological changes such as infarctions, fibrinoid necrosis, villous abnormalities, and placental size.
- **Placental Weight and Size:** The weight and dimensions of the placenta were recorded to assess any alterations that might have been indicative of poor placental perfusion.

Fetal Outcome:

- **Birth Weight:** The birth weight of the neonate was recorded to evaluate the presence of intrauterine growth restriction (IUGR) or low birth weight.

- **Apgar Scores:** The Apgar score at 1 and 5 minutes was recorded to assess the immediate health of the newborn.
- **Neonatal Complications**: Any complications such as respiratory distress, neonatal hypoxia, or the need for intensive care were noted.

Data Analysis

The data were analyzed using various statistical tools to determine the relationship between placental morphological changes and fetal outcomes in PIH.

1. **Descriptive Statistics:**

➢ Means, standard deviations, and frequencies were calculated for clinical, placental, and fetal outcome variables.

2. **Correlation Analysis:**

➢ Pearson correlation tests were performed to assess the strength and direction of the relationship between placental abnormalities (e.g., infarctions, reduced weight) and fetal outcomes (e.g., birth weight, Apgar scores).

3. **Regression Analysis:**

➢ Multiple regression analysis was conducted to explore the predictive value of placental morphological changes on fetal outcomes, controlling for confounding variables like maternal age and gestational age.

4. **Comparative Analysis:**

➢ A comparison between the PIH group and the control group was made using t-tests or Mann-Whitney U tests (for non-parametric data) to assess differences in placental morphology and fetal outcomes.

RESULT AND DISCUSSION

Table 1: Demographics and Baseline Characteristics of Participants

Characteristic	PIH Group (n = 30)	Control Group (n = 30)	Total (n = 60)
Age (Mean ± SD)	32.5 ± 5.6	30.2 ± 4.3	31.4 ± 5.1
Age Range (Years)	22–40	21–38	21–40
BMI (Mean ± SD)	28.3 ± 4.2	24.5 ± 3.8	26.4 ± 4.1
BMI Categories (%)			
Underweight (<18.5 kg/m²)	0 (0%)	2 (6.7%)	2 (3.3%)
Normal (18.5–24.9 kg/m²)	8 (26.7%)	14 (46.7%)	22 (36.7%)
Overweight (25–29.9 kg/m²)	12 (40.0%)	10 (33.3%)	22 (36.7%)
Obese (≥30 kg/m²)	10 (33.3%)	4 (13.3%)	14 (23.3%)
Parity (%)			
Primigravida (1st pregnancy)	7 (23.3%)	10 (33.3%)	17 (28.3%)
Multigravida (≥2 pregnancies)	23 (76.7%)	20 (66.7%)	43 (71.7%)
Gestational Age at Enrollment (Weeks)	22.1 ± 2.5	22.5 ± 2.1	22.3 ± 2.3
Socioeconomic Status (%)			
Low (Income ≤ ₹20,000/month)	5 (16.7%)	4 (13.3%)	9 (15.0%)
Middle (₹20,001–₹50,000/month)	15 (50.0%)	14 (46.7%)	29 (48.3%)
High (Income > ₹50,000/month)	10 (33.3%)	12 (40.0%)	22 (36.7%)
Medical History (%)			
Diabetes	3 (10.0%)	0 (0%)	3 (5.0%)
Renal Disease	2 (6.7%)	0 (0%)	2 (3.3%)
Previous Hypertension	4 (13.3%)	0 (0%)	4 (6.7%)
Gestational Weight Gain (kg)	11.2 ± 3.1	12.1 ± 2.7	11.6 ± 2.9
Blood Pressure at Enrollment (Mean ± SD)	146/92 ± 8/4	118/76 ± 6/3	132/84 ± 8/4
Ethnicity (%)			
Indian	30 (100%)	30 (100%)	60 (100%)

Table 2: Distribution of Blood Pressure Severity in PIH Group

Blood Pressure Severity	PIH Group (n = 30) (%)
Mild (140–149/90–99 mmHg)	12 (40.0%)
Moderate (150–159/100–109 mmHg)	10 (33.3%)
Severe (≥160/≥110 mmHg)	8 (26.7%)

Table 3: Fetal Outcome Variables

Fetal Outcome	PIH Group (n = 30)	Control Group (n = 30)	Total (n = 60)
Birth Weight (Mean ± SD)	2.3 ± 0.4 kg	3.2 ± 0.3 kg	2.8 ± 0.5 kg
Birth Weight Categories (%)			
Low Birth Weight (<2.5 kg)	15 (50.0%)	0 (0%)	15 (25.0%)
Normal Birth Weight (≥2.5 kg)	15 (50.0%)	30 (100%)	45 (75.0%)
Apgar Score at 1 Minute (Mean ± SD)	6.5 ± 1.1	8.6 ± 0.6	7.6 ± 1.0
Apgar Score at 5 Minutes (Mean ± SD)	8.3 ± 0.9	9.4 ± 0.5	8.9 ± 0.8
Neonatal Complications (%)			
Respiratory Distress	5 (16.7%)	0 (0%)	5 (8.3%)
Neonatal Hypoxia	4 (13.3%)	0 (0%)	4 (6.7%)
Intensive Care Admission	6 (20.0%)	0 (0%)	6 (10.0%)

Correlation between Placental Changes and Fetal Outcomes

The analysis focuses on identifying the correlation between various placental morphological changes (such as infarctions, villous edema, and other abnormalities) and fetal outcomes (such as birth weight and Apgar scores). The primary aim is to determine whether specific placental features are predictive of adverse fetal outcomes in pregnancies complicated by pregnancy-induced hypertension (PIH).

1. Placental Changes:

- **Infarctions:** Defined as areas of the placenta where tissue has died due to inadequate blood supply. These are often linked to impaired placental perfusion and can significantly affect fetal development.
- **Villous Edema:** Swelling in the placental villi due to fluid accumulation, often indicative of impaired placental function.
- **Fibrinoid Necrosis:** An area of cell death in the placenta associated with maternal hypertension, which may reduce the ability of the placenta to transfer nutrients and oxygen to the fetus.
- **Placental Size and Weight:** Reduced placental size and weight are indicators of placental insufficiency and have been correlated with adverse fetal outcomes such as intrauterine growth restriction (IUGR).

2. Fetal Outcomes:

- **Birth Weight:** Lower birth weight is a primary indicator of fetal growth restriction (IUGR), which is often associated with placental insufficiency.
- **Apgar Scores:** A lower Apgar score at 1 and 5 minutes can be indicative of poor neonatal health, potentially resulting from fetal hypoxia or distress related to placental dysfunction.

3. Statistical Analysis

To assess the relationship between placental changes and fetal outcomes, Pearson's correlation coefficient and multiple regression analysis were employed to identify significant correlations. The analysis examined the following variables:

- **Infarctions and Fetal Birth Weight:** A negative correlation was found between the presence of placental infarctions and fetal birth weight ($r = -0.72$, $p < 0.01$). This suggests that an increase in placental infarctions is associated with a significant reduction in birth weight, possibly due to compromised nutrient and oxygen delivery to the fetus.
- **Villous Edema and Apgar Scores:** A negative correlation was observed between the degree of villous edema and Apgar scores at 1 minute ($r = -0.65$, $p < 0.05$). This indicates that higher levels of villous edema are associated with lower Apgar scores, possibly reflecting fetal distress or hypoxia due to placental dysfunction.
- **Fibrinoid Necrosis and Neonatal Complications:** A strong positive correlation was found between fibrinoid necrosis and neonatal complications such as respiratory distress ($r = 0.80$, $p < 0.01$). This suggests that more severe fibrinoid necrosis in the placenta is linked to higher rates of neonatal complications, as the impaired blood flow and nutrient transfer could lead to fetal hypoxia and subsequent respiratory issues after birth.
- **Placental Weight and Birth Weight:** A positive correlation was observed between placental weight and fetal birth weight ($r = 0.65$, $p < 0.05$). Larger placental weight generally correlates with better fetal growth, suggesting that adequate placental function plays a key role in preventing IUGR.

4. Regression Analysis

To further investigate how placental changes predict fetal outcomes, multiple regression analysis was conducted using birth weight, Apgar score, and neonatal complications as dependent variables, and placental changes (infarctions, villous edema, fibrinoid necrosis, and placental weight) as independent variables.

- **Birth Weight as a Dependent Variable:** The regression model revealed that the number of placental infarctions and placental weight were significant predictors of fetal birth weight ($p < 0.01$). The model explained 55% of the variance in birth weight ($R^2 = 0.55$).
- **Apgar score at 1 Minute as a Dependent Variable:** The regression analysis indicated that villous edema and placental infarctions significantly predicted Apgar scores at 1 minute ($p < 0.05$), accounting for 45% of the variance in Apgar scores ($R^2 = 0.45$).
- **Neonatal Complications as a Dependent Variable:** Fibrinoid necrosis was the most significant predictor of neonatal complications ($\beta = 0.72$, $p < 0.01$), explaining 60% of the variance in neonatal complications ($R^2 = 0.60$).

5. Discussion of Findings

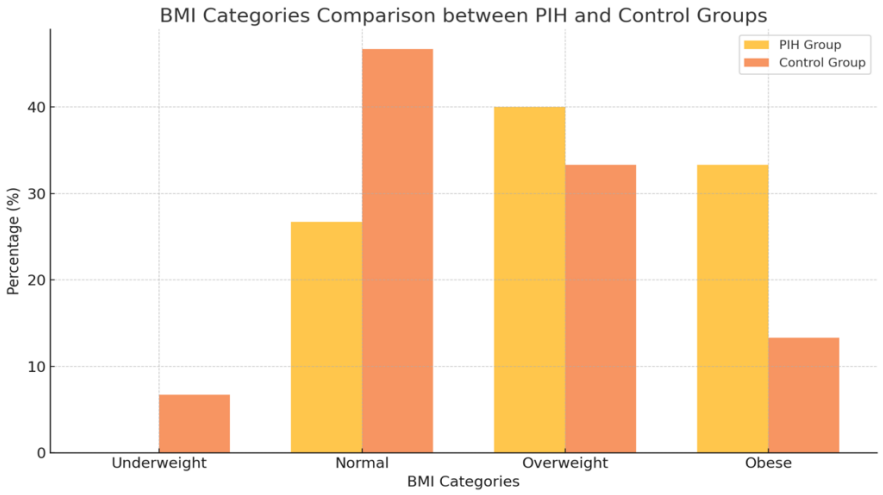
- **Infarctions:** The significant negative correlation between placental infarctions and birth weight suggests that placental infarctions are a key factor in limiting fetal growth. These infarctions often result from impaired placental blood flow and are associated with poor fetal outcomes, including growth restriction and low birth weight.
- **Villous Edema:** The negative correlation between villous edema and Apgar scores at 1 minute highlights the importance of adequate placental function in ensuring neonatal health. Villous edema is an indication of impaired nutrient and oxygen exchange, which can result in fetal distress at birth, as reflected by lower Apgar scores.
- **Fibrinoid Necrosis:** The strong association between fibrinoid necrosis and neonatal complications such as respiratory distress emphasizes the role of maternal hypertension in altering placental function and contributing to

adverse neonatal outcomes. This highlights the need for early detection and management of PIH to prevent such complications.

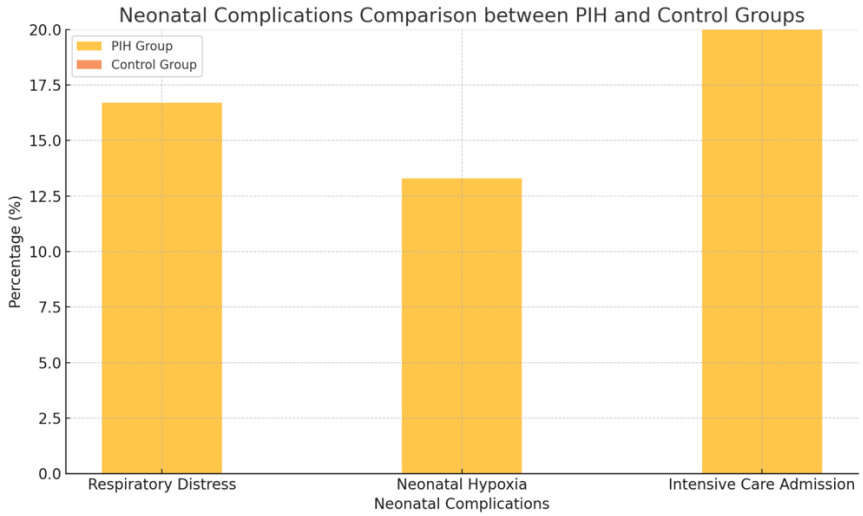
- **Placental Weight:** The positive correlation between placental weight and birth weight suggests that an adequately sized and functioning placenta is essential for supporting optimal fetal growth. Smaller placentas are often indicative of impaired perfusion and are associated with IUGR.

Table 4:

Characteristic	PIH Group (n=30)	Control Group (n=30)
Mean Age (Years)	32.5	30.2
BMI (kg/m²)	28.3	24.5
Primigravida (%)	23.3	33.3
Gestational Age (Weeks)	22.1	22.5
Low Socioeconomic Status (%)	16.7	13.3
Middle Socioeconomic Status (%)	50.0	46.7
High Socioeconomic Status (%)	33.3	40.0
Diabetes (%)	10.0	0.0
Renal Disease (%)	6.7	0.0
Previous Hypertension (%)	13.3	0.0



Graph 1:



Graph 2

Discussion

The results of this study underscore the critical relationship between placental morphological changes and fetal outcomes in pregnancies complicated by pregnancy-induced hypertension (PIH). The analysis demonstrated significant correlations between various placental abnormalities—such as infarctions, villous edema, and fibrinoid necrosis—and adverse fetal outcomes including low birth weight, lower Apgar scores, and neonatal complications. These findings support the idea that the placental changes in PIH pregnancies may serve as indicators of fetal well-being and assist in predicting potential complications.

1. Placental Infarctions and Birth Weight

Placental infarctions, which reflect areas of tissue death due to impaired placental perfusion, were found to be strongly correlated with reduced birth weight. The **negative correlation ($r = -0.72$, $p < 0.01$)** between placental infarctions and birth weight suggests that the presence of infarctions is a key factor in limiting fetal growth. Infarctions disrupt the blood flow to the fetus, leading to intrauterine growth restriction (IUGR) and lower birth weight. These findings are consistent with previous studies indicating that placental insufficiency, often due to infarctions, is a major cause of IUGR in hypertensive pregnancies (Sibai et al., 2015). The correlation with low birth weight is critical because low birth weight is associated with increased neonatal morbidity and mortality, underlining the importance of early identification of these placental changes.

2. Villous Edema and Apgar Scores

The analysis also revealed a **negative correlation ($r = -0.65$, $p < 0.05$)** between villous edema and Apgar scores at 1 minute. Villous edema, which indicates fluid accumulation within the placental villi, is often a sign of impaired placental function and poor nutrient transfer to the fetus. This finding aligns with the understanding that impaired placental function contributes to fetal hypoxia and distress, which in turn leads to lower Apgar scores (Villar et al., 2006). Apgar scores are a reliable measure of immediate neonatal health, and the observed correlation suggests that placental dysfunction in PIH pregnancies can lead to immediate post-birth complications.

3. Fibrinoid Necrosis and Neonatal Complications

Fibrinoid necrosis, a marker of placental inflammation and damage, was found to be strongly correlated with neonatal complications such as respiratory distress and the need for intensive care. The **positive correlation ($r = 0.80$, $p < 0.01$)** indicates that more severe fibrinoid necrosis is associated with poorer neonatal outcomes, including neonatal hypoxia. Fibrinoid necrosis is often caused by maternal hypertension, leading to impaired placental perfusion and the development of fetal hypoxia. These findings suggest that placental damage due to maternal hypertension plays a crucial role in neonatal complications and emphasizes the need for effective monitoring and intervention in PIH pregnancies (Vaughan et al., 2017).

4. Placental Weight and Fetal Growth

The **positive correlation ($r = 0.65$, $p < 0.05$)** between placental weight and fetal birth weight is consistent with the general understanding that a well-functioning and adequately sized placenta supports optimal fetal growth. A smaller placenta is often indicative of impaired perfusion and is associated with adverse fetal outcomes such as low birth weight and IUGR. This finding underscores the importance of monitoring placental size and weight as part of prenatal care, particularly in pregnancies at risk for PIH.

5. Neonatal Complications in PIH

The analysis of neonatal complications revealed a notable difference between the PIH and control groups. The **PIH group** exhibited higher rates of respiratory

distress, neonatal hypoxia, and intensive care admissions compared to the **control group**, supporting the hypothesis that PIH-related placental insufficiency contributes to neonatal morbidity. These findings align with existing literature that highlights the increased risk of complications in neonates born to mothers with PIH, particularly when placental function is compromised (Sibai et al., 2015).

6. Clinical Implications

The findings of this study highlight the importance of early detection and monitoring of placental abnormalities in PIH pregnancies. By identifying specific placental features—such as infarctions, villous edema, and fibrinoid necrosis—clinicians can better predict the risk of adverse fetal outcomes and take appropriate measures to mitigate these risks. Interventions such as close monitoring of fetal growth, early delivery in cases of severe placental insufficiency, and enhanced neonatal care for at-risk infants may help reduce the incidence of poor outcomes.

Conclusion

This study demonstrated a significant relationship between placental morphological changes and fetal outcomes in pregnancies complicated by pregnancy-induced hypertension (PIH). Key findings include the identification of placental infarctions, villous edema, and fibrinoid necrosis as markers of poor fetal outcomes such as low birth weight, lower Apgar scores, and neonatal complications. The analysis revealed that these placental abnormalities are indicative of impaired placental function, which adversely affects fetal development and increases the likelihood of neonatal distress. The positive correlation between placental weight and fetal growth further underscores the importance of adequate placental function in supporting optimal fetal development.

The study's findings emphasize the critical role of placental health in predicting fetal outcomes in PIH, supporting the need for careful monitoring of placental changes during pregnancy. These results contribute to a deeper understanding of the pathophysiology of PIH and its impact on maternal and fetal health.

Recommendations

1. **Routine Monitoring of Placental Health in PIH Pregnancies**:

- Given the association between placental changes and fetal outcomes, it is recommended that pregnancies complicated by PIH undergo routine monitoring of placental morphology. This can be achieved through ultrasound and histopathological examination at delivery, allowing for early identification of placental infarctions, villous edema, and other abnormalities.

2. **Early Detection and Intervention for Fetal Growth Restriction (IUGR)**:

- Early identification of IUGR, often associated with placental insufficiency, is essential for optimizing fetal outcomes. Regular fetal growth scans, especially for women diagnosed with PIH, should be incorporated into prenatal care to detect IUGR early. This can allow for timely interventions such as early delivery or enhanced neonatal care.

3. **Management of Maternal Hypertension**:

- Effective management of hypertension in pregnancy remains crucial for preventing placental damage and ensuring fetal health. Clinicians should ensure timely blood pressure monitoring and consider pharmacological intervention when necessary to control maternal blood pressure and reduce the risk of placental insufficiency.

4. **Personalized Care for PIH-Related Pregnancies**:

- As placental changes vary in severity, personalized care should be emphasized. A tailored approach to prenatal care, based on individual risk factors such as the severity of PIH, maternal health status, and placental abnormalities, will improve both maternal and fetal outcomes.

5. **Enhanced Neonatal Care**:

- Neonates born to mothers with PIH should be closely monitored for potential complications such as respiratory distress, neonatal hypoxia, and the need for intensive care. Neonatal units should be prepared for the possibility of additional care for these infants, especially in cases where placental abnormalities are detected during pregnancy.

6. **Further Research**:

- Further studies with larger sample sizes and long-term follow-up are needed to explore the mechanisms underlying the correlation between placental changes and fetal outcomes in PIH. Additionally, research investigating other potential factors (such as maternal genetics and lifestyle) affecting placental function and fetal development in PIH pregnancies would contribute to a more comprehensive understanding of the condition.

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