



ANALYSIS OF PLACENTAL PROFILE IN PREGNANCY WITH HYPERTENSION AND ITS RELATIONSHIP WITH THE CONDITION OF THE NEWBORN

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ABSTRACT

Hypertension in pregnancy (HDK) is one of the main complications that contribute to maternal and infant morbidity and mortality. This study aims to analyze the placental profile in pregnancies with hypertension and its relationship with the condition of the newborn. This study used an observational analytical design with a case-control approach. The sample consisted of 60 respondents consisting of 30 mothers with HDK (case group) and 30 mothers without hypertension (control group), selected using consecutive sampling technique. Data analysis was performed using t-test, Chi-square, and logistic regression with a significance level of $p < 0.05$. There were significant differences in placental profiles between the case and control groups, where the HDK group had lower weight (420 ± 55 vs 510 ± 60 grams; $p = 0.001$), diameter (16.5 ± 1.8 vs 18.2 ± 1.5 cm; $p = 0.002$), and thickness (2.1 ± 0.4 vs 2.8 ± 0.5 cm; $p = 0.001$). The incidence of infarction (60.0% vs 20.0% ; $p = 0.002$) and calcification (66.7% vs 26.7% ; $p = 0.001$) was higher in the HDK group. The condition of newborns in the HDK group showed a higher incidence of LBW (50.0% vs 20.0% ; $p = 0.015$), APGAR <7 (40.0% vs 16.7% ; $p = 0.032$), and asphyxia (43.3% vs 16.7% ; $p = 0.021$). Analysis showed that low placental weight (OR=3.8 ; $p = 0.008$), infarction (OR=4.5; $p = 0.004$), and calcification (OR=3.9; $p = 0.006$) were significantly associated with the condition of newborns. Placental profiles in hypertensive pregnancies experienced significant changes and were closely associated with the condition of newborns.

Keywords: BBLR; hypertension in pregnancy; hypoxia; neonatal placental profile

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INTRODUCTION

Hypertension in pregnancy (HDK) is one of the most serious obstetric complications and contributes significantly to maternal and infant morbidity and mortality worldwide. Globally, HDK occurs in approximately 5–10% of all pregnancies and is a leading cause of maternal death, contributing approximately 14% of total maternal deaths (Annisa et al., 2024). Furthermore, HDK also plays a significant role in increasing the risk of poor neonatal outcomes, such as prematurity, low birth weight (LBW), asphyxia, and even neonatal death (Podungge, 2023). Recent data show that babies born to mothers with hypertension have a two to three times higher risk of complications compared to normal pregnancies (Nurhidayati, 2026).

Pathophysiologically, HDK is closely related to impaired placental development and function. Failure of trophoblast invasion and spiral artery remodeling leads to uteroplacental insufficiency, which leads to hypoxia, oxidative stress, and chronic inflammation in placental tissue (Wibowo et al., 2023). This condition not only affects fetal growth but also causes various morphological and histopathological changes in the placenta, such as decreased placental weight, infarction, calcification, and vascular changes (Resia & Zubaidah, 2021). The placenta plays a central role as an organ that facilitates the exchange of nutrients and oxygen between the mother and fetus (Subani et al., 2020). Therefore, changes in the placental profile can be an important indicator in determining the condition of the newborn. Research shows that placental abnormalities are significantly associated with the incidence of low birth weight (LBW), low APGAR scores, and impaired neonatal adaptation (Yusnanda & Pratiwi, 2022). In fact, several recent studies confirm

that placental profile evaluation can be used as an early predictor of neonatal outcomes in high-risk pregnancies, including HDK (Louisa et al., 2024). Although the relationship between HDK and neonatal outcomes has been extensively researched, studies specifically linking placental profiles as key mediators of neonatal outcomes are still relatively limited, particularly in developing countries (Podungge et al., 2023). Most studies focus primarily on maternal clinical aspects or direct neonatal outcomes, without exploring placental changes as a key underlying mechanism (Safitri et al., 2021). This has led to a lack of comprehensive understanding of the pathophysiological pathways linking HDK to neonatal outcomes.

In Indonesia, including in Papua, HDK remains a serious challenge in maternal healthcare. However, data on placental characteristics in pregnancies with HDK and their relationship to newborn health are still very limited. In fact, understanding the placental profile is very important to support early detection, appropriate management, and prevention of more severe neonatal complications. Based on these problems, this study has high urgency because it attempts to fill the knowledge gap regarding the role of the placenta in determining infant outcomes in pregnancies with hypertension. By identifying placental profiles and analyzing their relationship with newborn conditions, it is hoped that this research can provide scientific contributions in developing early detection strategies and improving the quality of maternal and neonatal services. Therefore, this study aims to analyze the placental profile in pregnancies with hypertension and its relationship with the condition of the newborn.

METHOD

This study used an observational analytical design with a case-control approach to analyze the relationship between placental profiles in pregnancies with hypertension and newborn conditions. The research was conducted at Bhayangkara Hospital, Jayapura City, Papua, from April to July 2025. The research population included all mothers giving birth and their babies born at the hospital. The research sample was divided into two groups, namely the case group consisting of mothers with hypertension in pregnancy (HDK) and the control group, namely normal pregnant women without hypertension. The sampling technique used consecutive sampling in accordance with the inclusion criteria until the number of samples was met, with each group consisting of 30 respondents so that the total sample was 60 respondents. The inclusion criteria included mothers giving birth with a gestational age of ≥ 37 weeks, mothers with a diagnosis of HDK for the case group, mothers with normal pregnancies for the control group, placentas in intact condition and can be examined, and willingness to be respondents, while the exclusion criteria included mothers with comorbidities, multiple pregnancies, incomplete or damaged placentas, and incomplete infant data.

The variables in this study consisted of hypertension in pregnancy (HDK) as the independent variable and the condition of the newborn as the dependent variable, with the indicators measured including birth weight, APGAR score, and the incidence of asphyxia. In addition, placental profile acts as an intervening variable (mediator) including placental weight, placental diameter, placental thickness, and the presence of infarction and calcification. Operational definitions used include HDK, characterized by blood pressure $\geq 140/90$ mmHg after 20 weeks of gestation, low birth weight (LBW) if less than 2500 grams, and asphyxia if the APGAR score is less than 7 in the first minute. Placental profile is defined as the morphological characteristics of the placenta measured directly after delivery. The research instruments used included observation sheets to record maternal and infant characteristics, placental measuring instruments such as digital scales and measuring tapes, and medical record forms to obtain maternal and infant clinical data.

The research procedure began with the identification of mothers who met the criteria, then grouped respondents into case and control groups. Newborn data were obtained from medical records, while placental examinations were conducted immediately after delivery by weighing the placenta, measuring its diameter and thickness, and observing for infarction and calcification. All data were

then recorded in an observation sheet. Data analysis was performed using statistical software through several stages, namely univariate analysis to describe the frequency distribution of maternal characteristics, placental profiles, and newborn conditions, bivariate analysis using the Chi-square test for categorical data and the t-test or Mann-Whitney test for numerical data to see the relationship between HDK and placental profiles and placental profiles with newborn conditions, and multivariate analysis using logistic regression to determine the variables that most influence newborn conditions. The significance level in this study was set at $p < 0.05$.

RESULT

Table 1.
Respondent Characteristics (n=60)

Characteristics	Category	Case (HDK) n=30	%	Control n=30	%
Age	<20 years	2	6.7	3	10.0
	20–35 years	20	66.7	22	73.3
	>35 years	8	26.6	5	16.7
Parity	Primigravida	14	46.7	12	40.0
	Multigravida	16	53.3	18	60.0

Table 2.
Comparison of Placental Profiles between Case and Control Groups

Variables	Cases (HDK) Mean ± SD	Control Mean ± SD	p-value
Placental weight (grams)	420 ± 55	510 ± 60	0.001
Diameter (cm)	16.5 ± 1.8	18.2 ± 1.5	0.002
Thickness (cm)	2.1 ± 0.4	2.8 ± 0.5	0.001

Table 3.
Description of Placental Abnormalities in Case and Control Groups

Placental Abnormalities	Case (HDK) n=30	%	Control n=30	%	p-value
Infarction (+)	18	60.0	6	20.0	0.002
Calcification (+)	20	66.7	8	26.7	0.001

Table 4.
Condition of Newborns in the Case and Control Groups

Variables	Case (HDK) n=30	%	Control n=30	%	p-value
LBW (<2500 gr)	15	50.0	6	20.0	0.015
APGAR <7	12	40.0	5	16.7	0.032
Asphyxia	13	43.3	5	16.7	0.021

Table 5.
Relationship between Placental Profile and Newborn Condition

Placental Variables	BBL Condition	OR (95% CI)	p-value
Low placental weight	Low birth weight	3.8 (1.4–10.2)	0.008
Placental infarction	Asphyxia	4.5 (1.6–12.3)	0.004
Classification	APGAR <7	3.9 (1.5–10.5)	0.006

The results showed significant differences in placental profiles between the group of mothers with gestational hypertension and the control group. The mean placental weight, diameter, and thickness in the case group were lower than in the control group ($p < 0.05$). Furthermore, placental infarction and calcification were more common in the case group. The condition of newborns in the case group also showed worse outcomes compared to the control group, marked by an increase in the incidence of LBW, low APGAR scores, and asphyxia ($p < 0.05$). Association analysis showed that changes in placental profile, such as low placental weight, infarction, and calcification, were significantly associated with newborn outcomes. Low-weight placentas had a 3.8 - fold increased risk of low birth weight (LBW), while placental infarction increased the risk of asphyxia by up to 4.5 times.

DISCUSSION

The results of this study indicate that mothers with gestational hypertension (GHD) have significant changes in placental profile compared to normal pregnancies, characterized by decreased placental

weight, diameter, and thickness, as well as increased incidence of infarction and calcification. These changes are closely associated with poorer newborn outcomes, such as increased incidence of low birth weight (LBW), low Apgar scores, and asphyxia. These findings reinforce the central role of the placenta as a major mediator between maternal conditions and neonatal outcomes.

Pathophysiologically, HDK is initiated by the failure of trophoblast invasion in early pregnancy which causes inadequate remodeling of the spiral arteries. This condition results in decreased uteroplacental perfusion, limiting the supply of oxygen and nutrients to the fetus. Consequently, chronic hypoxia occurs in the placenta, triggering oxidative stress and an inflammatory response (Ministry of Health of the Republic of Indonesia, 2023) . This hypoxia then causes structural damage to the placenta, manifested as decreased placental weight, infarction, and calcification, as found in this study (Adam et al., 2023) .

The decrease in placental weight and size in the HDK group reflects a reduced capacity for maternal-fetal exchange. A smaller placenta has a limited surface area for oxygen and nutrient diffusion, thus directly contributing to fetal growth restriction and low birth weight (LBW) (Safitri & Djaiman, 2021) . The results of this study indicate that low placental weight increases the risk of low birth weight by up to 3.8 times, indicating a strong link between placental insufficiency and suboptimal fetal growth.

In addition, the high incidence of placental infarction in the HDK group indicates more severe perfusion disorders. Placental infarction is an area of tissue necrosis caused by reduced blood flow, which directly reduces the placenta's ability to maintain fetal oxygenation. In this study, placental infarction was shown to increase the risk of asphyxia by up to 4.5 times. This can be explained by the fact that chronic hypoxia that occurs during pregnancy can progress to acute hypoxia during delivery, making it difficult for the baby to adapt after birth (Muthmainah, 2021). The greater prevalence of placental calcification in the HDK group also indicates a degenerative process due to prolonged oxidative stress. Extensive calcification can disrupt placental elasticity and vascular function, further impairing uteroplacental perfusion. This condition contributes to low Apgar scores in newborns, reflecting impaired physiological adaptation immediately after birth (Raihan & Iqbal, 2025).

This sequence of changes indicates a clear pathophysiological pathway, namely HDK causing placental insufficiency → chronic hypoxia → damage to placental structure → impaired oxygen supply to the fetus → poor neonatal outcomes . In other words , the placenta acts as a primary mediator connecting maternal conditions with those of the newborn. Therefore, evaluating the placental profile is not only important as a retrospective indicator, but also has the potential to be an early predictive tool for the risk of neonatal complications (Fitri et al., 2023) The results of this study align with various international studies that indicate that impaired placental perfusion in HDK is significantly associated with an increased incidence of low birth weight (LBW), asphyxia, and prematurity (Subani et al., 2020) . Other studies have also shown that changes in placental morphology, such as infarction and calcification, are correlated with fetal hypoxia and poor neonatal outcomes (Yusnanda & Pratiwi, 2022) . This further strengthens the argument that a placenta-based approach can provide a more comprehensive understanding than solely examining maternal or neonatal factors.

The clinical implications of this study are very important in obstetric and neonatology practice. Early identification of changes in placental profile in mothers with HDK can help health workers in stratifying risk and determining more appropriate interventions, such as close fetal monitoring, determining the timing of delivery, and readiness for neonatal resuscitation. Furthermore, postpartum placental examination can also be used as an evaluation tool for planning future pregnancies. However, this study has limitations, including the relatively limited sample size and

the lack of in-depth placental histopathology. Therefore, further research is recommended to integrate histopathology and placental biomarker analysis to obtain a more comprehensive picture of the mechanisms involved.

CONCLUSION

This study shows that there are significant differences in placental profiles between pregnancies with hypertension and normal pregnancies, which are characterized by a decrease in placental weight, diameter, and thickness as well as an increase in the incidence of infarction and calcification. These changes in placental profile have been shown to be significantly associated with neonatal outcomes, with mothers with gestational hypertension having a higher risk of delivering babies with low birth weight, low Apgar scores, and asphyxia. Pathophysiologically, gestational hypertension causes impaired uteroplacental perfusion, leading to chronic hypoxia, triggering structural and functional damage to the placenta. This condition disrupts the supply of oxygen and nutrients to the fetus, ultimately impacting poor neonatal outcomes. Thus, the placental profile acts as an important mediator connecting maternal conditions with the condition of the newborn.

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