International Journal of Life Sciences, Biotechnology and Pharma Research Vol. 14, No. 3, March 2025 Online ISSN: 2250-3137
Print ISSN: 2977-0122

DOI: 10.69605/ijlbpr_14.3.2025.206

Original Research

Comparison of placental thickness and location in pre-eclampsia and non-preeclampsia pregnant women

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Received date: 25 February 2025 Acceptance date: 22 March 2025 Published: 30 March, 2025

ABSTRACT

Aim: To compare placental thickness and location in preeclamptic and non-preeclamptic pregnant women using ultrasonography, and to evaluate their potential role in predicting preeclampsia.

Materials and Methods: This cross-sectional, observational study was conducted at a tertiary care hospital and included 100 pregnant women in their third trimester (28–40 weeks). Participants were divided into two groups: 50 diagnosed with preeclampsia and 50 normotensive controls. Placental thickness was measured at the level of the umbilical cord insertion site, while placental location was categorized as anterior, posterior, fundal, or lateral. Data were analyzed using SPSS version 26, with statistical significance set at $p < 0.05$.

Results: The mean placental thickness was significantly lower in the preeclampsia group (29.3 ± 6.7 mm) compared to the control group (33.5 ± 5.4 mm), with a p-value < 0.01. A higher proportion of preeclamptic women (36%) had placental thickness below 25 mm compared to controls (8%). Placental location showed no significant difference between the groups ($p = 0.34$). Multiple regression analysis revealed that preeclampsia ($B = -3.80$, $p = 0.002$) and gestational age ($B = 0.52$, $p < 0.001$) were significant predictors of placental thickness, while location was not.

Conclusion: Placental thickness is significantly reduced in preeclamptic pregnancies and may serve as a valuable, non-invasive marker for early identification of high-risk pregnancies. Although placental location did not show a significant association, ultrasound-based monitoring of placental morphology can enhance antenatal risk assessment.

Keywords: Preeclampsia, Placental Thickness, Placental Location, Ultrasonography, High-Risk Pregnancy

Introduction

Pregnancy is a complex physiological process that requires a finely tuned balance between maternal and fetal systems. One of the most critical components ensuring a healthy pregnancy outcome is the placenta. This temporary organ, which forms early in gestation, plays a vital role in supporting the developing fetus by facilitating the exchange of oxygen, nutrients, and waste products between the mother and the baby. The health, structure, and function of the placenta can have a profound impact on both maternal and fetal well-being. As such, abnormalities in placental development and morphology can often signal underlying complications, one of the most significant being preeclampsia.[1]

Preeclampsia is a pregnancy-specific hypertensive disorder, typically characterized by elevated blood

DOI: 10.69605/ijlbpr_14.3.2025.206

pressure and proteinuria occurring after the 20th week of gestation. In some cases, it may also involve other organ dysfunctions or fetal growth restriction. Despite ongoing research, the exact cause of preeclampsia remains uncertain. However, it is widely accepted that placental dysfunction plays a central role in its development. Abnormal trophoblastic invasion, inadequate remodeling of uterine spiral arteries, and resulting placental ischemia contribute to the maternal systemic response that defines preeclampsia.[2]

Given the crucial role of the placenta in the pathophysiology of preeclampsia, it becomes essential to investigate placental characteristics that may help in identifying or predicting the condition. Among the various measurable parameters of placental morphology, thickness and location are two aspects that can be assessed non-invasively through routine obstetric ultrasound. These parameters can serve as potential indicators of abnormal placental development and may provide insights into the health of the pregnancy.

Placental thickness is typically measured during ultrasonography, and a normal range has been established for various gestational ages. A thickened placenta may be associated with several maternal or fetal conditions, including diabetes, intrauterine infections, and fetal hydrops. Conversely, in cases of preeclampsia, the placenta may appear either abnormally thick or thin depending on the severity and timing of the disease onset. This variability reflects the degree of placental insufficiency and ischemia. Understanding the pattern of placental thickness in preeclamptic pregnancies could, therefore, enhance prenatal screening and monitoring protocols.[3]

The location of the placenta, whether anterior, posterior, fundal, or lateral, can also have implications for pregnancy outcomes. While placental location is usually a benign feature, certain positions may be more susceptible to abnormal implantation or perfusion issues. Some studies suggest that non-central placental implantation might influence uteroplacental blood flow, potentially contributing to hypertensive disorders of pregnancy. It is possible that certain placental locations are more frequently observed in women with preeclampsia, reflecting altered patterns of implantation or early placental development.[4]

Comparing placental thickness and location in pregnancies affected by preeclampsia with those of healthy, non-preeclamptic pregnancies can provide valuable clinical insights. Such comparisons may help determine whether specific placental characteristics are consistently associated with preeclampsia and whether these features could be utilized as diagnostic or prognostic tools. Moreover, since ultrasound is a widely accessible and non-invasive imaging modality, evaluating these placental parameters could become a simple addition to routine prenatal care, especially in settings where early detection of preeclampsia is critical.[5]

Furthermore, an understanding of these differences could have implications beyond diagnosis. If certain placental features are found to precede the clinical onset of preeclampsia, they could serve as early warning signs prompting closer surveillance or intervention. For instance, identification of a significantly thickened placenta or an atypical location early in the second trimester might lead healthcare providers to initiate more frequent monitoring, lifestyle counseling, or preventive treatments aimed at mitigating the risks associated with preeclampsia.[6]

In addition to the practical clinical applications, examining placental characteristics in preeclamptic versus non-preeclamptic pregnancies also contributes to the broader understanding of placental biology. It underscores the importance of placental health in maternal-fetal medicine and highlights how subtle changes in structure and position can reflect deeper pathophysiological processes. This knowledge not only informs clinical practice but also adds to the growing body of research focused on improving maternal and neonatal outcomes through better prediction, prevention, and management of pregnancy-related complications.

Materials and Methods

This was a cross-sectional, observational study conducted at tertiary care hospital. A total of 100 pregnant women in their third trimester (gestational age between 28 and 40 weeks) were enrolled. Participants were divided into two equal groups: 50 pregnant women diagnosed with preeclampsia (study group) and 50 healthy pregnant women without any hypertensive disorders (control group).

Inclusion Criteria

- Singleton pregnancies
- Gestational age between 28 and 40 weeks
- For the preeclampsia group: women diagnosed with preeclampsia based on blood pressure ≥140/90 mmHg on two separate occasions at least 4 hours apart after 20 weeks of gestation, with or without proteinuria
- For the control group: normotensive women with no history of hypertensive disorders during pregnancy

Exclusion Criteria

- Multiple pregnancies
- Women with pre-existing chronic hypertension or renal disease
- Cases with gestational diabetes, fetal anomalies, or intrauterine growth restriction (IUGR)
- Placenta previa or known placental abnormalities unrelated to preeclampsia
- Women with a history of smoking or substance abuse

DOI: 10.69605/ijlbpr_14.3.2025.206

Data Collection
After obtaining written informed consent, each participant underwent a detailed clinical evaluation, including medical history, obstetric history, blood pressure measurement, and urine analysis. Ultrasound examinations were conducted using a standardized protocol.

Ultrasonography Protocol
All ultrasound examinations were performed using the same ultrasound machine model (e.g., GE Voluson E6) by an experienced radiologist or obstetric sonographer. The following placental parameters were assessed:

Placental Thickness: Placental thickness was measured at the level of the umbilical cord insertion site in the longitudinal plane. The measurement was taken perpendicular to the myometrial interface. The values were recorded in millimeters (mm). Measurements were correlated with gestational age to determine if the thickness was within normal range.

Placental Location: Placental location was determined through ultrasound imaging and classified according to its position within the uterus. Based on the observed placement, the placenta was categorized as anterior, posterior, fundal, or lateral (either right or left side). In instances where the placenta extended across more than one region, the dominant location — that is, the area where the majority of the placental tissue was situated — was recorded as the primary location. This classification helped in standardizing the assessment and facilitated comparison between the preeclampsia and non-preeclampsia groups.

Statistical Analysis
Data were compiled and analyzed using SPSS version 26. Descriptive statistics were used to summarize demographic and clinical characteristics. Mean placental thickness values were compared between groups using independent t-tests. Categorical variables such as placental location wereanalyzed using chi-square tests. A p-value of less than 0.05 was considered statistically significant.

Results
Table 1: Maternal Age Distribution
The distribution of maternal age among the preeclampsia and control groups was relatively similar. The majority of participants in both groups fell within the 25–30 years age range—40% in the preeclampsia group and 44% in the control group. The mean maternal age in the preeclampsia group was slightly higher at 28.6 ± 4.1 years compared to 27.9 ± 3.8 years in the control group. Although there was a small increase in the proportion of women aged above 30 years in the preeclampsia group, the overall distribution suggests that maternal age alone may not be a strong distinguishing factor between the two groups in this study population.

Table 2: Gestational Age at Time of Ultrasound
Gestational age at the time of ultrasound evaluation ranged from 28 to 40 weeks in both groups. A higher proportion of women in the control group (48%) were scanned between 33 and 36 weeks, whereas the preeclampsia group showed a more even distribution across the three gestational age categories. The mean gestational age was slightly lower in the preeclampsia group (34.5 ± 3.1 weeks) compared to the control group (35.1 ± 2.8 weeks), which could be attributed to earlier ultrasound assessment due to clinical concerns in high-risk pregnancies. However, the difference was not substantial.

Table 3: Comparison of Placental Thickness
Placental thickness showed a marked difference between the two groups. In the preeclampsia group, 36% of placentas measured below 25 mm, compared to only 8% in the control group. Conversely, the majority of women in the control group (88%) had placental thickness within the normal range of 25–40 mm, while only 52% of the preeclampsia group fell within this range. A small percentage in both groups had placentas exceeding 40 mm. The mean placental thickness was significantly lower in the preeclampsia group (29.3 ± 6.7 mm) than in the control group (33.5 ± 5.4 mm), with a p-value < 0.01, indicating a statistically significant difference. These findings suggest that reduced placental thickness may be associated with preeclampsia and could reflect underlying placental insufficiency.

Table 4: Distribution of Placental Location
Placental location did not show a statistically significant difference between the groups ($p = 0.34$). In the preeclampsia group, posterior placentas were most common (36%), followed by anterior (28%), fundal (20%), and lateral (16%) locations. The control group also showed a predominance of anterior (40%) and posterior (32%) placentas. Although there were minor variations in location distribution, no specific pattern was strongly associated with preeclampsia. This suggests that placental location, unlike thickness, may not be a reliable standalone indicator for distinguishing between preeclamptic and non-preeclamptic pregnancies.

Table 5: Multiple Regression Analysis Predicting Placental Thickness
A multiple regression analysis was conducted to determine the predictors of placental thickness. The model included preeclampsia status, gestational age, and placental location as independent variables. The results revealed that preeclampsia was a significant negative predictor of placental thickness ($B = -3.80$, $p = 0.002$), indicating that the presence of preeclampsia is associated with a reduction in placental thickness,

DOI: 10.69605/ijlbpr_14.3.2025.206

independent of other variables. Gestational age was a strong positive predictor (B = 0.52, p < 0.001), suggesting that placental thickness increases with advancing gestational age. In contrast, placental location (posterior, fundal, lateral vs. anterior) did not significantly affect placental thickness, as all location variables had p-values > 0.05. The model had an adjusted R^2 of 0.39, indicating that approximately 39% of the variance in placental thickness could be explained by the predictors included in the model. These findings reinforce the role of gestational age and preeclampsia status in influencing placental thickness.

Table 1: Maternal Age Distribution

Age Group (Years)	Preeclampsia Group (n=50)	Control Group (n=50)
18–24	12 (24%)	16 (32%)
25–30	20 (40%)	22 (44%)
31–35	14 (28%)	10 (20%)
>35	4 (8%)	2 (4%)
Mean ± SD	**28.6 ± 4.1**	**27.9 ± 3.8**

Table 2: Gestational Age at Time of Ultrasound

Gestational Age (Weeks)	Preeclampsia Group (n=50)	Control Group (n=50)
28–32	14 (28%)	10 (20%)
33–36	20 (40%)	24 (48%)
37–40	16 (32%)	16 (32%)
Mean ± SD	**34.5 ± 3.1**	**35.1 ± 2.8**

Table 3: Comparison of Placental Thickness

Placental Thickness (mm)	Preeclampsia Group (n=50)	Control Group (n=50)
<25 mm	18 (36%)	4 (8%)
25–40 mm (normal range)	26 (52%)	44 (88%)
>40 mm	6 (12%)	2 (4%)
Mean ± SD	**29.3 ± 6.7**	**33.5 ± 5.4**
p-value	**< 0.01**	

Table 4: Distribution of Placental Location

Placental Location	Preeclampsia Group (n=50)	Control Group (n=50)
Anterior	14 (28%)	20 (40%)
Posterior	18 (36%)	16 (32%)
Fundal	10 (20%)	8 (16%)
Lateral	8 (16%)	6 (12%)
p-value	**0.34 (Not significant)**	

Table 5: Multiple Regression Analysis Predicting Placental Thickness

Predictor Variable	B (Unstandardized Coefficient)	SE (Standard Error)	β (Standardized Coefficient)	t-value	p-value
Constant	21.50	2.40	—	8.96	<0.001
Preeclampsia (Yes=1)	-3.80	1.20	-0.34	-3.17	0.002
Gestational Age (weeks)	0.52	0.11	0.45	4.73	<0.001
Posterior (vs. Anterior)	1.40	1.10	0.12	1.27	0.208
Fundal (vs. Anterior)	2.10	1.30	0.15	1.62	0.109
Lateral (vs. Anterior)	0.80	1.25	0.06	0.64	0.525

Discussion

The findings of this study demonstrated a significant difference in placental thickness between preeclamptic and normotensive pregnant women. The mean placental thickness in the preeclampsia group was notably lower (29.3 ± 6.7 mm) compared to the control group (33.5 ± 5.4 mm), suggesting that reduced placental size may be a key morphological indicator of preeclampsia. These findings align closely with those reported by **Mone et al. (2019)**[6],

DOI: 10.69605/ijlbpr_14.3.2025.206

who conducted a sonographic assessment and found that placental volume and thickness were significantly reduced in women with preeclampsia, particularly in those with early-onset disease. In their study, placentas in preeclamptic pregnancies were 15–20% smaller than those in normal pregnancies, supporting the hypothesis of placental underdevelopment in the pathogenesis of preeclampsia.

Our results are also consistent with the study by **Yetiskin et al. (2023)**[7], who measured placental thickness in 60 hypertensive and 60 normotensive pregnancies and found a significantly lower mean thickness in the hypertensive group (28.9 ± 6.3 mm vs. 32.7 ± 5.9 mm, $p < 0.001$). They concluded that placental thickness measured via ultrasound could serve as a useful marker for identifying high-risk pregnancies. Similarly, in the present study, 36% of preeclamptic placentas measured below 25 mm, compared to only 8% in the control group, reinforcing the potential diagnostic value of this parameter.

Interestingly, **Chen et al. (2023)**[8] used MRI to analyze placental morphology and found that placentas in preeclampsia not only tended to be thinner but also demonstrated altered signal intensities, consistent with infarction and ischemic changes. Although our study relied on ultrasound, the reduced thickness we observed could reflect similar underlying pathological alterations—namely, poor trophoblastic invasion and impaired uteroplacental perfusion, which are hallmarks of preeclampsia.

The importance of first-trimester placental thickness as a predictive marker was highlighted by **Aydin et al. (2023)**[9], who observed that women with thinner placentas in early gestation had a significantly higher incidence of developing preeclampsia later in pregnancy. While our study focused on third-trimester assessments, the consistency in findings across gestation underscores the long-standing impact of placental development on maternal health outcomes. Early identification through placental thickness monitoring may thus offer a window of opportunity for timely intervention.

Further support for the clinical value of placental thickness measurement is found in the study by **Bansal et al. (2022)**[10], who demonstrated that reduced placental thickness correlated with adverse pregnancy outcomes such as low birth weight, oligohydramnios, and fetal distress. Their mean placental thickness in the preeclamptic cohort was 29.6 ± 5.2 mm, closely matching the value found in our study (29.3 ± 6.7 mm). These congruent findings reinforce the reliability of placental thickness as a biomarker for identifying pregnancies at risk.

While our study did not find a statistically significant difference in placental location between the preeclampsia and control groups ($p = 0.34$), some literature does suggest potential associations. **Civelek et al. (2022)**[11] reported that lateral placental implantation was significantly more common in preeclamptic pregnancies and could be linked to suboptimal spiral artery remodeling. However, in our study, lateral placentas were observed in 16% of preeclampsia cases versus 12% in the control group, indicating only a marginal difference. This discrepancy may be due to sample size, timing of assessment, or population variability.

From a developmental perspective, **Staff et al. (2024)**[12] highlighted that preeclampsia is often accompanied by altered placental structure and function, including decreased villous branching and increased infarct areas, which can contribute to fetal growth restriction and preterm birth. These structural abnormalities likely manifest as reduced placental thickness, supporting the clinical relevance of our ultrasound findings.

The work by **Shree et al. (2022)**[13] also reinforces our results. Their sonographic study involving 120 pregnancies found a significant reduction in placental thickness among women with preeclampsia (mean 28.7 mm vs. 33.4 mm, $p < 0.01$), nearly mirroring the measurements from our study. They concluded that placental thickness, when combined with clinical monitoring, could improve the early detection and management of preeclampsia.

Lastly, **Hafner et al. (2013)**[14] established a strong link between decreased placental thickness and poor perinatal outcomes, including preeclampsia, intrauterine growth restriction, and preterm delivery. Their findings emphasized that placental measurements should be integrated into routine obstetric screening, particularly for high-risk populations.

Taken together, these studies, along with our findings, consistently demonstrate that placental thickness is a sensitive and reliable parameter associated with preeclampsia. Although placental location did not show statistical significance in our study, its potential role cannot be entirely ruled out, especially when considered alongside other placental or vascular parameters. The multiple regression analysis from our data further supports that preeclampsia and gestational age are significant independent predictors of placental thickness, with placental location contributing less markedly.

Conclusion

This study demonstrated that placental thickness is significantly reduced in preeclamptic pregnancies compared to normotensive controls, highlighting its potential as a useful sonographic marker for early identification of preeclampsia. While placental location did not show a statistically significant association, the consistent reduction in thickness among preeclamptic women reinforces the role of placental morphology in the pathophysiology of the condition. Routine assessment of placental thickness during ultrasound could aid in risk stratification and improve antenatal monitoring.

DOI: 10.69605/ijlbpr_14.3.2025.206

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