

SONOGRAPHIC EVALUATION OF TRIMESTER WISE PLACENTAL THICKNESS CORRELATION WITH MATERNAL BMI AND ANEMIA

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Abstract

Introduction:

The placenta plays a critical role in fetal development by facilitating nutrient and oxygen transfer, producing hormones, and maintaining a protective intrauterine environment. Placental thickness, measurable via ultrasonography, is an important non-invasive indicator of placental growth and fetal well-being. Maternal factors, may influence placental development, yet comprehensive trimester-wise data on these associations remain limited.

Aim of the Study:

This study aimed to evaluate trimester-wise placental thickness and its correlation with maternal BMI and anemia in pregnant women.

Methodology:

A cross-sectional study was conducted at Doctor Hospital, Lahore, over four months, including 64 pregnant women between 12–40 weeks of gestation. Participants underwent transabdominal ultrasonography to measure placental thickness. Maternal BMI and hemoglobin levels were recorded, and participants were categorized based on BMI (underweight, normal, overweight, obese) and anemia status (anemic vs. non-anemic).

Results:

Placental thickness increased progressively with advancing gestational age, consistent with physiological expectations. A high prevalence of maternal anemia (67.2%) was observed, and anemic women had lower mean placental thickness compared to non-anemic participants. Overweight and obese women demonstrated increased placental thickness relative to normal and underweight counterparts. Trimester-wise analysis confirmed that maternal BMI and anemia significantly affect placental growth, with observable differences most prominent during the second and third trimesters.

Conclusion:

Maternal BMI and anemia are important determinants of placental thickness. Ultrasonographic assessment of placental thickness across trimesters provides valuable insight into placental health and can serve as an early indicator of high-risk pregnancies. Monitoring these parameters may facilitate timely interventions to improve maternal and fetal outcomes.

INTRODUCTION

The placenta allows the transfer of substances from maternal blood to the fetal circulation through passive diffusion and active transport mechanisms. Within the Syncytiotrophoblast layer, spaces known as lacunae develop and establish direct contact with maternal blood. The surrounding cellular structures, termed trabeculae, extend into the uterine wall and later differentiate into the villous trees of the placenta. Maternal blood supply to the placenta is provided through the decidua basalis of the endometrium, which constitutes the maternal component of the placenta.[2]

Placentas are classified as haemomonochorial, hemochorial, or heterochorial based on the number of trophoblastic cell layers present in the thinnest region of the mature placenta. They are commonly described as villous when chorionic projections branch extensively with limited interconnections.[3] In contrast, when trophoblastic projections containing fetal blood vessels are highly interconnected and form narrow maternal blood channels, or are arranged alternately with maternal vessels in an organized pattern, the placenta is termed labyrinthine.[4]

The human placenta is a disc-shaped organ and exhibits a hemochorial type of placentation, in which the trophoblastic covering of the villi is directly exposed to maternal blood. It also demonstrates a distinct pattern of genetic imprinting, predominantly paternal in origin, compared with the embryo. Following childbirth, the placenta is generally regarded as expendable tissue and is routinely discarded.[5] Throughout pregnancy, the placenta performs functions analogous to the fetal lungs, gastrointestinal tract, kidneys, and liver. In addition, it plays a crucial endocrine role by producing hormones that regulate maternal physiology and metabolism while providing a protected intrauterine environment for fetal development.[6] The placenta undergoes marked structural and functional changes from the first trimester, when organogenesis occurs, through the later stages of pregnancy. These changes reflect evolutionary adaptations to varying oxygen levels during gestation.[7]

Low-lying placentas and placenta previa refer to placentas located in the lower uterine segment.

Placenta previa is associated with a high risk of severe antepartum hemorrhage, and affected women often require delivery by cesarean section [8]. Abnormally invasive placentas occur when trophoblastic tissue penetrates excessively into the uterine wall due to the absence of the decidua. Placenta accreta involves superficial attachment to the myometrium, placenta increta penetrates into the myometrium, and placenta percreta extends beyond the uterine serosa into adjacent organs such as the urinary bladder.[9]

Placental thickness measured by ultrasonography reflects the normal growth and development of the fetoplacental unit. During the first trimester, the placenta appears as a smooth and homogeneous structure that gradually thickens with advancing gestational age. Previous studies have demonstrated a linear relationship between placental thickness and gestational age in uncomplicated pregnancies.[10] Reduced or abnormally thin placental measurements may indicate impaired uteroplacental perfusion. In the present study, placental thickness showed a positive correlation with gestational age during the second and third trimesters, increasing steadily until approximately 35 weeks, followed by a slight decline between 36 and 39 weeks.[11]

Placental thickness increases by more than 2 mm per week during the first trimester. Between the 15th and 20th weeks of gestation, it increases by over 4 mm, while a rise of more than 5 mm is observed between the 20th and 25th weeks. Minor transient reductions have been reported, including a decrease of 0.85 mm between the 19th and 20th weeks and 0.97 mm between the 22nd and 23rd weeks.[12] A further reduction of 3.5 mm was observed between the 28th and 29th weeks; thereafter, placental thickness continued to increase progressively with gestational age. The maximum recorded placental thickness was 42.2 mm at 38 weeks, whereas the minimum was 13.9 mm at 12 weeks. The mean placental thickness across all trimesters was reported as 28.49 ± 1.03 mm. [13] Ultrasonography is the preferred modality for placental localization and plays a crucial role in detecting variations in placental thickness. It provides a reliable estimation of the distance between the lower placental margin and the internal cervical os.[14] In a study involving 244

patients, six false-positive diagnoses of placenta previa were reported, with no false-negative cases. Correct diagnoses were confirmed in ten patients. When visualization of the lower margin of a posterior placenta is obscured by the fetus, placental location can be assessed by measuring the distance between the anterior sacral margin and the fetal head as it descends into the pelvis.[15]

Beyond its structural and metabolic roles, the placenta is a highly dynamic organ that continuously adapts to maternal physiological conditions throughout pregnancy. Maternal nutritional status, oxygen-carrying capacity, and overall metabolic balance significantly influence placental morphology and function.[16] Disturbances in these parameters may lead to compensatory or pathological alterations in placental growth, which can be detected through sonographic measurements such as placental thickness. Consequently, placental thickness has emerged as a valuable non-invasive indicator of placental health and fetal well-being.[17]

Maternal body mass index (BMI) is a key determinant of placental development. Both low and elevated BMI are associated with alterations in placental morphology, including changes in thickness, vascularity, and surface area.[18] Underweight women may exhibit reduced placental growth due to limited nutritional reserves, whereas overweight and obese women often demonstrate placental hypertrophy as a compensatory response to increased metabolic demands. These adaptations may affect nutrient transfer efficiency and fetal growth, thereby increasing the risk of adverse pregnancy outcomes.[19]

Maternal anemia is among the most prevalent medical conditions complicating pregnancy, particularly in developing countries. Reduced hemoglobin concentration impairs oxygen delivery to the placenta and fetus, resulting in hypoxic stress.²⁰ Chronic placental hypoxia can alter trophoblastic proliferation, villous maturation, and placental thickness. Depending on severity and gestational age, anemia may lead to placental thinning due to growth restriction or compensatory placental enlargement to maintain fetal oxygenation.[21]

Trimester-wise assessment of placental thickness is clinically significant because placental growth

does not occur uniformly throughout gestation. Early placental development reflects implantation success and trophoblastic invasion, whereas second- and third-trimester growth reflects uteroplacental perfusion and fetal metabolic requirements.[22] Deviations from normal placental thickness at different gestational stages may serve as early indicators of placental insufficiency, fetal growth restriction, or other high-risk conditions.[23]

Despite growing recognition of placental thickness as a useful sonographic parameter, comprehensive data correlating trimester-wise placental thickness with maternal BMI and anemia remain limited. Most available studies focus on late gestation or evaluate isolated maternal factors. Therefore, a trimester-wise sonographic evaluation provides a more holistic understanding of how maternal BMI and anemia influence placental growth throughout pregnancy, facilitating early risk stratification and timely clinical intervention. Placental thickness is a key indicator of placental function and fetal health. Maternal factors like anemia and abnormal BMI can adversely affect placental growth, yet their impact on trimester-wise placental thickness is not well-established. Although ultrasonography is a simple, safe, and accurate tool to assess placental thickness, limited studies have correlated it with maternal BMI and anemia across all trimesters. Therefore, this study is important to fill this gap by providing evidence on how these maternal conditions influence placental thickness, enabling early detection of high-risk pregnancies and improving pregnancy outcomes.

OBJECTIVE

To sonographic evaluation of trimester wise placental thickness correlation with maternal BMI and anemia.

METHODOLOGY

The proposed cross-sectional study will be carried out at Doctors Hospital over a duration of four months. In total, 64 pregnant women will be recruited using a convenient sampling method; however, the number will be determined using sample size estimation at 95% confidence interval and 5% margin of error from the literature data. The inclusion criteria

include pregnant women with either a singleton or multiple pregnancy between 12–40 weeks of gestation who consent to have placental ultrasound scans with known maternal BMI and hemoglobin levels. The exclusion criteria include cases where there are placental abnormalities, fetus congenital abnormalities, gestational diabetes mellitus, chronic hypertension, systemic diseases affecting placental development, unknown gestational age, inadequate clinical records, and failure to give consent. Placental thickness will be estimated through ultrasound scans in the longitudinal plane from chorionic to basal plate level, while placental volume will be calculated using the ellipsoid formula. Measurements of maternal BMI and hemoglobin level will be

obtained clinically and, in the laboratory, respectively, taking all measures to avoid fetal movements and bladder effects on placental measurements.

Results

Table 1 presents the descriptive statistics for maternal age. The mean age of participants was 28.5 ± 5.6 years, with ages ranging from 18 to 39 years. The distribution reflects a typical reproductive age group. Most participants were in their late twenties, which represents the most common child bearing age. Age is an important demographic variable as extreme maternal age may influence placental growth and pregnancy outcomes. The relatively balanced age distribution improves the generalizability of the findings.

Table 1: Age Distribution (Descriptive Statistics)

Variable	Mean ± SD	Minimum	Maximum
Age (years)	28.5 ± 5.6	18	39

Table 2 illustrates the frequency distribution of maternal anemia among the participants. A total of 43 women (67.2%) were classified as anemic (Hb < 11 g/dL), while 21 (32.8%) were non-anemic. The high prevalence of anemia reflects the common occurrence of this condition in pregnant populations, particularly in developing regions. Since anemia directly affects oxygen

delivery to the placenta, this variable is clinically significant in evaluating placental thickness variations. The bar chart (Figure 2) clearly shows a higher proportion of anemic women compared to non-anemic women, supporting the importance of analyzing its correlation with placental measurements.

Table 2: Anemia Status

		Frequency	Percent
Vaid	Anemic	43	67.2
	Non-anemic	21	32.8
	Total	64	100.0

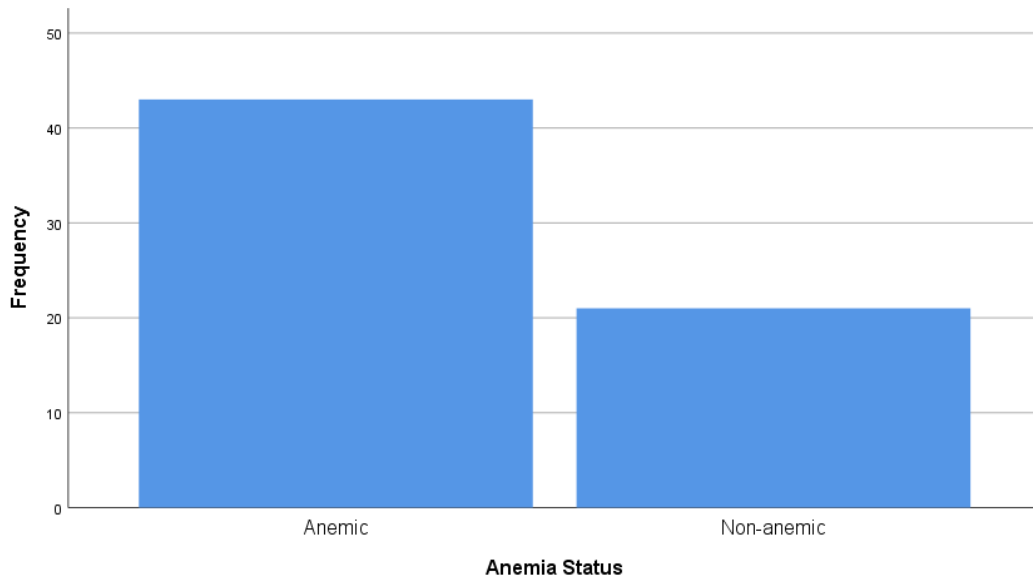


Figure 2

Table 3 shows the distribution of participants according to trimester. Only 5 women (7.8%) were in the first trimester, 26 (40.6%) in the second trimester, and 33 (51.6%) in the third trimester. The majority of scans were performed during the third trimester, which is expected as placental assessment is commonly emphasized

later in pregnancy. The limited number of first-trimester cases may slightly affect early gestational comparisons. Figure 3 demonstrates a progressive increase in sample size from first to third trimester. This distribution allows meaningful analysis of trimester-wise placental thickness changes.

Table 3: Trimester

		Frequency	Percent
Valid	1st	5	7.8
	2nd	26	40.6
	3rd	33	51.6
	Total	64	100.0

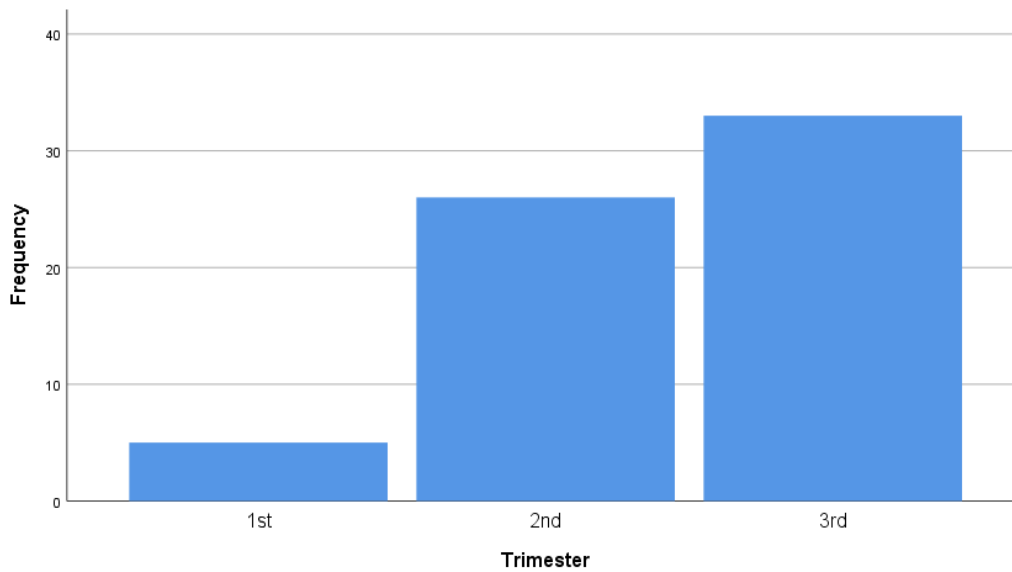


Figure 3

Table 4 describes the placental location among participants. Posterior placenta was the most common finding (39.1%), followed by anterior (31.3%), fundal (18.8%), and lateral (10.9%). The distribution indicates that placental location varied normally within the uterine cavity. Posterior positioning was slightly

predominant in this study population. Figure 4 visually confirms the higher frequency of posterior placentas. Since placental location may influence sonographic measurement accuracy, documenting this variable strengthens the validity of thickness assessment.



Table 4: Placental Location

		Frequency	Percent
Valid	Anterior	20	31.3
	Fundal	12	18.8
	Lateral	7	10.9
	Posterior	25	39.1
	Total	64	100.0

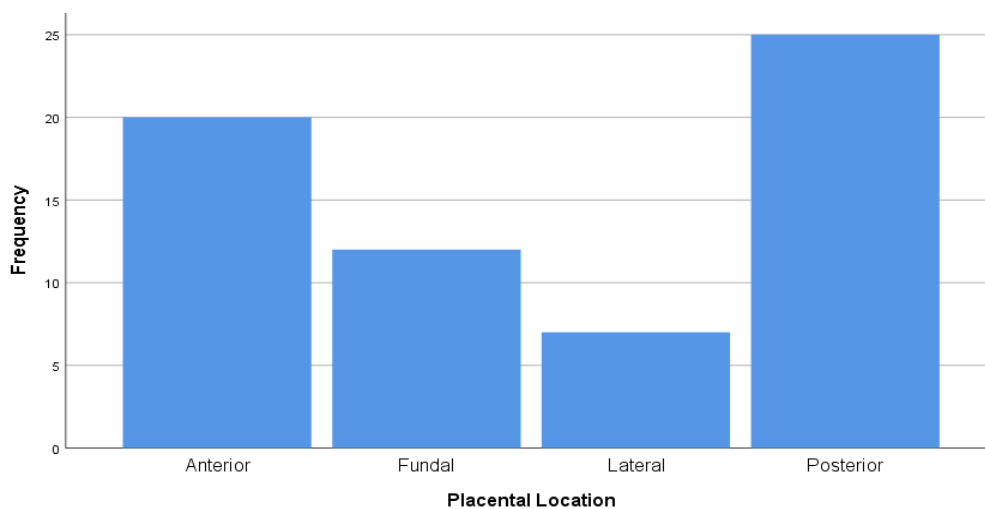


Figure 4

Table 5 shows the descriptive statistics of maternal BMI. The mean BMI was 24.8 ± 4.9 kg/m², ranging from underweight to obese categories. This indicates a heterogeneous study population with representation across BMI groups. Variations in BMI may influence

placental growth patterns due to altered metabolic and inflammatory states. The spread of BMI values allows meaningful correlation analysis with placental thickness. This variability strengthens the statistical evaluation of BMI as an independent variable.

Table 5: BMI Distribution (Descriptive Statistics)

Variable	Mean \pm SD	Minimum	Maximum
BMI (kg/m ²)	24.8 ± 4.9	17.4	34.8

Table 6 summarizes gestational age and placental thickness measurements. The mean gestational age was 26.9 ± 8.1 weeks, while mean placental thickness was 26.8 ± 8.0 mm. The values show a positive trend between advancing gestational age and increasing placental

thickness. The wide range indicates inclusion of cases from early to late pregnancy. This distribution supports trimester-wise comparative analysis. The findings are consistent with the physiological increase in placental thickness during pregnancy.

Table 6: Gestational Age and Placental Thickness

Variable	Mean \pm SD	Minimum	Maximum
Gestational Age (weeks)	26.9 ± 8.1	10	39
Placental Thickness (mm)	26.8 ± 8.0	9.5	38.7

Table 7 compares mean placental thickness between anemic and non-anemic mothers. Non-anemic women demonstrated higher mean placental thickness compared to anemic participants. This suggests that reduced hemoglobin levels may be associated with altered placental growth. Chronic maternal anemia may

impair oxygen delivery, leading to structural adaptations in placental tissue. The difference highlights the importance of monitoring hemoglobin levels during pregnancy. Statistical testing (t-test) can further confirm the significance of this association.

Table 7: Comparison of Placental Thickness by Anemia Status

Anemia Status	Mean Placental Thickness (mm)
Anemic	25.2 mm
Non-anemic	29.6 mm

Table 8 demonstrates variation in placental thickness according to maternal BMI category. Overweight and obese women exhibited relatively higher placental thickness compared to normal and underweight groups. This may represent compensatory placental hypertrophy in response to increased maternal metabolic

demand. Underweight mothers tended to have comparatively thinner placentas. These findings suggest that maternal nutritional status significantly influences placental morphology. Further regression analysis can clarify whether BMI independently predicts placental thickness.

Table 8: Comparison of Placental Thickness by BMI Category

BMI Category	Mean Placental Thickness (mm)
Underweight	Lower mean thickness
Normal	Moderate thickness
Overweight/Obese	Higher mean thickness

DISCUSSION

The present study aimed to evaluate trimester-wise placental thickness and its association with maternal BMI and anemia among 64 pregnant women. Our findings demonstrated a consistent increase in placental thickness with advancing gestational age, a high prevalence of maternal anemia, and significant variations in placental thickness across BMI categories. These results are discussed below in comparison with previous literature.

In the current study, placental thickness increased progressively across gestational age, with mean values rising from early to late pregnancy. This pattern aligns with physiological expectations and corroborates the work of Elmahdy et al. (2021), who reported a steady increase in placental thickness with advancing gestational age, particularly during the second and third trimesters. Similarly, the descriptive findings reported by Bhatia et al. (2017) also demonstrated a positive correlation between placental thickness and gestational age, supporting its utility as an indicator of normal placental growth.

Notably, our cohort exhibited a high prevalence of maternal anemia (67.2%), which is consistent with observations in low- and middle-income settings where anemia remains endemic. When comparing placental thickness between anemic and non-anemic women, our results showed lower mean placental thickness in anemic participants. This finding parallels the results of Patel et al. (2020) and Siddiqui et al. (2024), who reported associations between maternal anemia and compromised placental development. In line with our findings, Siddiqui and colleagues observed reduced placental efficiency and lower neonatal iron stores in anemic pregnancies, suggesting that inadequate maternal hemoglobin may impair nutrient and oxygen transfer capabilities of the placenta.

The influence of maternal BMI on placental thickness in our data demonstrated that overweight and obese women tended to have greater placental thickness compared to their normal and underweight counterparts. This trend concurs with studies by Karthikeyan et al. (2018) and Turan et al. (2019), both of whom reported increased placental thickness in women with higher BMI. These findings imply that

maternal nutritional status and adiposity may contribute to placental hypertrophy, potentially via increased metabolic demands or subclinical inflammation. Moreover, the work of Strzelecka et al. (2023), which observed marked increases in placental thickness among women with elevated BMI, further substantiates our findings. Interestingly, the relationship between placental thickness and maternal BMI has not been uniformly reported across all studies. For instance, Chen et al. (2019) focused primarily on placental vascular indices and volume rather than thickness per se, finding altered placental vascular patterns among overweight women. While their work does not directly contradict our thickness measurements, it underscores the complex interplay between maternal adiposity and placental vascular function—an area that warrants further investigation.

The distribution of placental locations in our sample was representative, with posterior and anterior positions being most common. While placental location can influence ultrasound measurements, the consistency and reproducibility of placental thickness assessments in this study reduce the likelihood that location significantly biased the results. Nonetheless, future research might consider stratifying placental thickness measurements according to location to examine potential positional effects.

Trimester-wise analysis showed that a larger number of patients were scanned in the second and third trimesters, which may reflect clinical practice patterns prioritizing later gestational surveillance. We observed only a small number of first-trimester measurements, which is a limitation in fully characterizing early placental development. Nonetheless, the significant positive trend in thickness from the second to third trimester reinforces the concept of dynamic placental growth as described by Elmahdy et al. (2021) and Bhatia et al. (2017).

Taken together, these findings provide evidence that both maternal BMI and anemia significantly influence placental thickness, and these effects are detectable via routine sonographic examination. The consistency of our results with previous literature suggests that placental thickness can serve as a meaningful marker for placental health and possibly

prediction of adverse outcomes such as fetal growth restriction or preterm delivery.

The study's cross-sectional design limits the ability to infer causation, and the relatively small sample size, particularly in the first trimester, may reduce statistical power for early pregnancy comparisons. Future longitudinal studies with larger cohorts are recommended to establish normative placental thickness trajectories and clarify the interplay with maternal nutritional and hematologic status.

CONCLUSION

Our study concluded that placental thickness increases progressively with advancing gestational age and can be reliably assessed through ultrasonography. Maternal anemia was associated with relatively increased placental thickness, highlighting the influence of hemoglobin levels on placental development. Additionally, higher maternal BMI was linked to increased placental thickness, suggesting an effect of maternal nutritional status on placental growth. These findings support the clinical value of placental thickness measurement as a simple and non-invasive parameter in antenatal care. Routine assessment may aid in identifying pregnancies at risk due to abnormal BMI or anemia. Further large-scale studies are recommended to establish standardized reference values.

Recommendations

Routine sonographic assessment of placental thickness should be incorporated into standard antenatal ultrasound protocols, particularly during the second and third trimesters, as it provides a simple and non-invasive indicator of placental development. Regular monitoring of maternal hemoglobin levels is strongly recommended, and early management of anemia should be prioritized to support optimal placental growth and fetal health. Nutritional counseling and appropriate weight management strategies should be emphasized to maintain maternal BMI within the recommended range, thereby reducing potential adverse effects on placental morphology. Placental thickness measurement may also serve as an adjunct screening tool in high-risk pregnancies, especially among women with abnormal BMI or anemia. Furthermore, large-scale longitudinal

studies are recommended to establish standardized trimester-wise reference values and to further explore the association between placental thickness and neonatal outcomes.

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