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Placental morphology and its association with fetomaternal outcomes: A prospective observational study

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Abstract
Introduction: The placenta plays an important role in pregnancy by facilitating nutrient and oxygen exchange between mother and fetus. Abnormal placental morphology is associated with adverse maternal and fetal outcomes, including hypertensive disorders, fetal growth restriction, and preterm birth. This study aimed to assess the relationship between placental characteristics (weight, thickness, diameter) and fetomaternal outcomes in Indian pregnant women.
Methods: A prospective observational study was conducted among 150 pregnant women (gestational age ≥34 weeks) at Sri Siddhartha Medical College and Hospital. Placental parameters (weight, thickness, diameter, ultrasound-measured thickness) were recorded, along with maternal (hypertension, diabetes, anemia) and fetal outcomes (birth weight, fetal growth restriction, neonatal intensive care unit admission). Statistical analysis was performed using SPSS v25.0.
Results: The mean placental weight was 446.1g, thickness 4.3cm, and diameter 17.8cm. Pregnancy-induced hypertension (16%) and gestational diabetes (13.3%) were associated with reduced placental dimensions ($p<0.05$). Fetal growth restriction (26.7%), neonatal intensive care unit admission (38%), and preterm birth (25.3%) were significantly more frequent in cases with lower placental weight (<400g), thickness (<2.5cm), and diameter (<18cm) ($p<0.001$). Anemia (12%) correlated with smaller placental size ($p=0.02$). Doppler studies indicated insufficiency in 28.7% of cases, though histopathology revealed no abnormalities.
Conclusion: Placental morphology significantly influences maternal and fetal outcomes. Smaller placental dimensions were associated with higher risks of hypertension, anemia, fetal growth restriction, and preterm birth. Comprehensive placental assessment may help predict adverse pregnancy outcomes and guide clinical management.

Keywords: Placental morphology, fetal growth restriction, placental insufficiency, maternal outcomes.

Introduction

The placenta is a unique feto-maternal organ that secretes hormones and molecules essential for the maintenance of pregnancy and acts as a critical connection between the mother and foetus by trafficking nutrients, oxygen and metabolic wastes [1, 2]. It is indispensable for foetal growth and sustains essential physiological roles typically performed by somatic organs postnatally. The site of implantation, which determines placental positioning and placental thickness directly influences placental perfusion and can consequently affect the maternal and perinatal outcomes [3]. Pregnancy complications, such as hypertensive disorders, gestational diabetes, and multiple pregnancies, significantly influence placental structure and function, as evident from both macroscopic and microscopic alterations [4]. Abnormal placental development is often associated with complications like early childbirth, inadequate foetal weight gain, restricted growth of the foetus, hypertensive disorders, twin birth and stillbirth. The primary obstetric causes of maternal mortality (MM) encompass severe bleeding, such as postpartum haemorrhage (PPH) and other factors. Additionally, hypertensive disorders of pregnancy, particularly preeclampsia and eclampsia, along with maternal infections or sepsis, significantly contribute to maternal deaths [5]. Globally, an estimated 3-4 million stillbirths and 3 million neonatal deaths occur each year, with 97-99% reported in LMIC [6]. Improved intrapartum monitoring and timely care could prevent over 40% of these stillbirths.

Around 75% of neonatal deaths occur within the first 7 days of birth, with approximately 1 million newborns dying within 24 hours of birth. Premature birth, birth complications (asphyxia and trauma), neonatal infections, and congenital anomalies remain the commonest causes of neonatal mortality, collectively contributing to nearly 40% of under-5 deaths [7].

Pre-eclampsia is associated with distinct placental pathologies, characterized predominantly by increased maternal vascular malperfusion and reduced inflammatory lesions. In pre-eclamptic pregnancies, maternal vascular malperfusion is observed in 57.3% of cases compared to 37.1% in normotensive pregnancies, with histological abnormalities identified in 61.3% of placentas, more than the 45.0% in pregnancies without pre-eclampsia. Outcomes such as stillbirth and preterm birth further underscore the association, with maternal vascular malperfusion present in 79.9% of pre-eclamptic stillbirths and 49.7% of preterm live births. Inflammatory lesions such as chorioamnionitis and funisitis, are less common in pre-eclamptic placentas, suggesting an association between vascular and inflammatory mechanisms in this condition [8]. A study identified significant placental pathologies associated with LBW babies, indicating chronic placental insufficiency. Gross examination revealed reduced placental weight and dimensions, with irregular placental shape in 26% of cases and paracentral umbilical cord insertion in 54%. Common gross lesions included infarctions (56%), subchorionic fibrin deposition (64%), and intervillous fibrin deposition (52%). Histopathological analysis demonstrated cytotrophoblast hyperplasia (52%), stromal fibrosis (48%), fibrinoid necrosis (100%), increased syncytial knots (32-68%), and hypovascular villi (44%). These abnormalities collectively indicate impaired placental perfusion and chronic insufficiency as key contributors to LBW outcomes. The findings emphasize the value of detailed placental examination for understanding the pathogenesis of LBW and guiding future obstetric care [9].

Placental pathology in congenital heart disease (CHD) cases is marked by lower placental weight and abnormalities indicative of impaired placentation, such as altered vascular and angiogenic gene expression. These findings are similar to the features observed in placentas from cases of preeclampsia (PE) and intrauterine growth restriction (IUGR), suggesting a shared origin in abnormal trophoblast invasion and placental insufficiency. Molecular analyses reveal altered protein and RNA expression in vascular developmental pathways, impacting placental vascularization and potentially foetal organ development. Hypoxic stress during early embryogenesis and hemodynamic effects of CHD may drive epigenetic changes, leading to angiogenic imbalances and placental dysfunction. Reduced placental blood flow, as evidenced by impaired umbilical venous blood volume, further corroborates compromised placental function in CHD cases. These abnormalities underscore the critical interplay between placental pathology and foetal outcomes [10]. Increase in the mother's during pregnancy shows a + correlation with increased placental dimensions, specifically length, breadth, and surface area, in cases of complete placenta previa, highlighting its potential impact on placental size and nutrient transfer capacity [11].

The placenta and umbilical cord serve as crucial indicators for predicting adverse foetal outcomes. As a key determinant of maternal and perinatal mortality, the placenta significantly influences foetal growth, with placental insufficiency often implicated in adverse pregnancy outcomes. Complications such as hypertensive disorders or gestational diabetes manifest both macroscopically and microscopically in the placenta, underscoring its role in prenatal care. Comprehensive sonographic assessment, gross evaluation, and histopathological examination of the placenta provide critical information for obstetricians and neonatologists alike. Despite its importance, there is a paucity of literature addressing these associations among Indian pregnant women, soliciting a detailed prospective study to explore these relationships. Henceforth this study aims to assess the feto-maternal outcomes affected by placental morphology in terms of placental weight, thickness, surface area; foetal and maternal outcomes in terms of placental morphology, ultrasound and histopathology.

Methodology

A hospital-based prospective observational study was conducted among pregnant women aged 18-40 years admitted to the Department of Obstetrics and Gynecology at Sri Siddhartha Medical College and Hospital, Tumakuru, over 18 months from April 2023 to September 2024. The study included women with a gestational age above 34 weeks who delivered either vaginally or via cesarean section, while excluding those with multiple pregnancies, retained placenta, morbidly adherent placenta, or unwillingness to participate. The sample size was 150, calculated using the formula $n = Z^2p(1 - p)/m^2$, where $n = 150$, $Z = 1.96$ (95% confidence level), $p = 50\%$ (proportion of the population), and $m = 8\%$ (margin of error). A convenience sampling method was employed, with participants enrolled based on inclusion and exclusion criteria.

Following approval from the Institutional Scientific and Ethics Committee, the study protocol was explained to participants, and informed consent was obtained. Each woman underwent a detailed clinical examination and history assessment to confirm eligibility. Data on maternal demographic and clinical parameters, placental characteristics, and neonatal outcomes were recorded. The placenta was collected immediately after delivery, drained of blood, washed under running tap water, and trimmed of membranes before examination. Sonographic evaluation was performed before birth, followed by gross and histopathological examination of all placentas, regardless of delivery mode.

Placental weight was measured using a digital baby weighing scale after cutting the umbilical cord approximately 5 cm from its insertion site. Surface area was calculated using the formula $\pi \times Dl \times Ds / 4$, where $\pi = 3.14$, Dl is the longest diameter, and Ds is the shortest diameter. Placental thickness was measured at the center using a graduated needle in millimeters. Any abnormalities, such as infarction, calcification, or hemorrhage, were noted. Ultrasonography was performed on all women beyond 34 weeks, with placental thickness assessed near the umbilical cord insertion site using pulsed Doppler evaluation of the umbilical artery, middle cerebral artery, ductus venosus, and uterine arteries. Histopathological analysis involved microscopic examination of at least six placental tissue blocks per case.

Data on placental weight, thickness, surface area, and ultrasonographic and histopathological findings were collected. The data were entered into Microsoft Excel 2010, cleaned for accuracy, and analyzed using SPSS version 25.0. Continuous variables were summarized as mean ± standard deviation or median with interquartile range, while categorical data were presented as counts and percentages. Statistical significance was assessed using the independent t-test, Mann-Whitney U test, or Chi-square test, with a p-value < 0.05 considered significant. A pre-formulated semi-structured questionnaire was used for data collection.

Results

The study population was primarily young women, with 44.7% aged 18-25 years, 30.7% aged 26-30 years, and 22.6% aged 31-35 years. Obstetric history revealed 43.3% were gravida 2, while 38.0% were gravida 1. Parity distribution showed 51.3% were nulliparous, 40.7% had one previous delivery, and only 8.0% had two prior deliveries. Regarding reproductive outcomes, 53.3% had no live births, 41.3% had one live birth, and 5.3% had two live births. Abortion history indicated 81.3% had no abortions, 10.7% had one abortion, and 6.7% had two abortions. Stillbirths were uncommon, with 95.3% reporting none and 4.7% reporting one previous stillbirth. Delivery modes were evenly distributed, with 50.0% undergoing cesarean section, 46.7% having vaginal deliveries, and 3.3% requiring instrumental delivery. Fetal Doppler ultrasound findings showed 28.7% with insufficiency and 71.3% within normal limits. (Table 1)

The mean gestational age was 37.4 weeks (SD = 1.5), ranging from 34.2 to 40 weeks. Placental measurements showed a mean thickness of 4.3 cm (SD = 0.6; range: 2.4-5.9 cm) and a mean weight of 446.1 grams (SD = 92.0; range: 216-710 grams). The average placental diameter measured 17.8 cm (SD = 3.8), with values spanning from 11 cm to 27 cm. Ultrasound assessment revealed a mean placental thickness of 37.8 mm (SD = 4.4), with measurements between 29 mm and 51 mm. Neonatal parameters demonstrated a mean birth weight of 2690.5 grams (SD = 552.7), ranging from 1300 grams to 4260 grams. The average birth length was 46.4 cm (SD = 1.7), with values from 43 cm to 51 cm, while head circumference averaged 34.1 cm (SD = 1.2), ranging between 31.5 cm and 37 cm. APGAR scores indicated good neonatal status, with mean values of 7.5 (SD = 0.7; range: 5-8) at 1 minute and 8.5 (SD = 0.7; range: 6-9) at 5 minutes. (Table 2)

Among the participants, pregnancy-induced hypertension affected 15.3% of mothers, while 12.7% developed gestational diabetes and 11.3% had anemia. Less common complications included placental abruption (1.3%), postpartum hemorrhage (2.7%), fever (5.3%), and premature rupture of membranes (6%). No cases of sepsis occurred. For fetal outcomes, 26.7% showed growth restriction, 38% required NICU admission, and 25.3% were preterm deliveries. Regarding birth weight, 30% of newborns weighed under 2.5 kg. Importantly, no intrauterine deaths were reported in any cases. (Figure 1)

When examining ultrasound-measured placental thickness, fetal growth restriction was most prevalent in the thinnest placentas (<35mm: 77.7%) ($p<0.001$). Similarly, NICU admission rates were highest in the thinnest placentas (86.1% at <35mm, $p<0.001$), while preterm birth showed a graded decrease from 52.7% to 0% across thickness categories ($p<0.001$). Interestingly, low birth weight (<2.5kg) demonstrated an inverse U-shaped relationship, affecting 16.6%, 89.6%, and 100% of cases from thinnest to thickest placentas respectively ($p<0.001$). Placental weight showed similar associations, with fetal growth restriction decreasing from 75.6% in the lightest placentas (<400g) to just 2.0% in the heaviest (>500g, $p<0.001$). NICU admissions followed this pattern (90.2% vs 22.0%, $p<0.01$), as did preterm birth rates (51.2% vs 6.0%, $p<0.001$). All infants from placentas weighing >400g had low birth weight, suggesting a threshold effect. For placental thickness measurements, the most dramatic differences emerged in the thinnest placentas (<2.5cm), where 100% of cases showed growth restriction and required NICU admission, compared to just 13% and 25% respectively in the thickest group (>4.0cm, $p<0.001$). Preterm birth showed a similar gradient (100% vs 18%, $p=0.002$). Placental diameter measurements revealed that smaller placentas (<18cm) had significantly higher rates of growth restriction (50.7% vs 3.1% in largest group, $p<0.001$), NICU admission (54.9% vs 34.3%), and preterm birth (40.8% vs 6.2%). (Table 3)

We observed that pregnancy-induced hypertension was most prevalent in the thinnest placentas (<35mm: 41.6%) compared to intermediate (35-45mm: 6.6%) and thickest (>45mm: 12.5%) groups ($p<0.001$). Gestational diabetes showed the opposite pattern, with 100% prevalence in the thickest placentas versus just 2.7% in the thinnest ($p<0.001$). Similarly, anemia was significantly more common in thinner placentas (27.7% at <35mm vs 0% at >45mm, $p<0.001$). When examining placental weight, hypertension decreased progressively from 26.8% in the lightest (<400g) to 6.0% in the heaviest (>500g) placentas ($p=0.02$), while diabetes showed a striking increase from 2.4% to 32.0% across the same weight categories ($p<0.001$). Placental thickness measurements demonstrated particularly dramatic differences, with both hypertension and anemia affecting 100% of women with the thinnest placentas (<2.5cm) compared to just 8.0% and 8.2% respectively in the thickest group (>4.0cm, $p<0.001$). For placental diameter, hypertension was most common with smaller diameters (23.9% at <18cm vs 6.3% at 18-21cm, $p=0.02$), while diabetes showed the opposite pattern (43.7% at >21cm vs 1.4% at <18cm, $p<0.001$). (Table 4)

Table 1: Demographic and obstetric characteristics of the study participants (N=150)

Variables	Frequency (n)	Proportion (%)
Age (years)		
18-25	67	44.7
26-30	46	30.7
31-35	34	22.6
36-40	3	2.0
Gravida		
1	57	38.0

2	65	43.3
3	22	14.7
4	6	4.0
Parity		
0	77	51.3
1	61	40.7
2	12	8.0
Live Birth		
0	80	53.3
1	62	41.3
2	8	5.3
Abortion		
0	122	81.3
1	16	10.7
2	10	6.7
3	2	1.3
Still Birth		
0	143	95.3
1	7	4.7
Mode of Delivery		
Vaginal	70	46.7
Instrumental	5	3.3
Cesarean	75	50.0
Fetal Doppler (USG)		
Insufficiency	43	28.7
Within normal limits	107	71.3

Table 2: Placental and neonatal characteristics of the study participants (N=150)

Variables	Mean	SD	Minimum	Maximum
Placental Characteristics				
Gestational age (weeks)	37.4	1.5	34.2	40
Placental thickness (cm)	4.3	0.6	2.4	5.9
Placental weight (gm)	446.1	92.0	216	710
Placental diameter (cm)	17.8	3.8	11	27
USG Placental thickness (mm)	37.8	4.4	29	51
Neonatal Characteristics				
Birth weight (gm)	2690.5	552.7	1300	4260
Birth length (cm)	46.4	1.7	43	51
Head circumference (cm)	34.1	1.2	31.5	37
APGAR - 1 min	7.5	0.7	5	8
APGAR - 5 min	8.5	0.7	6	9

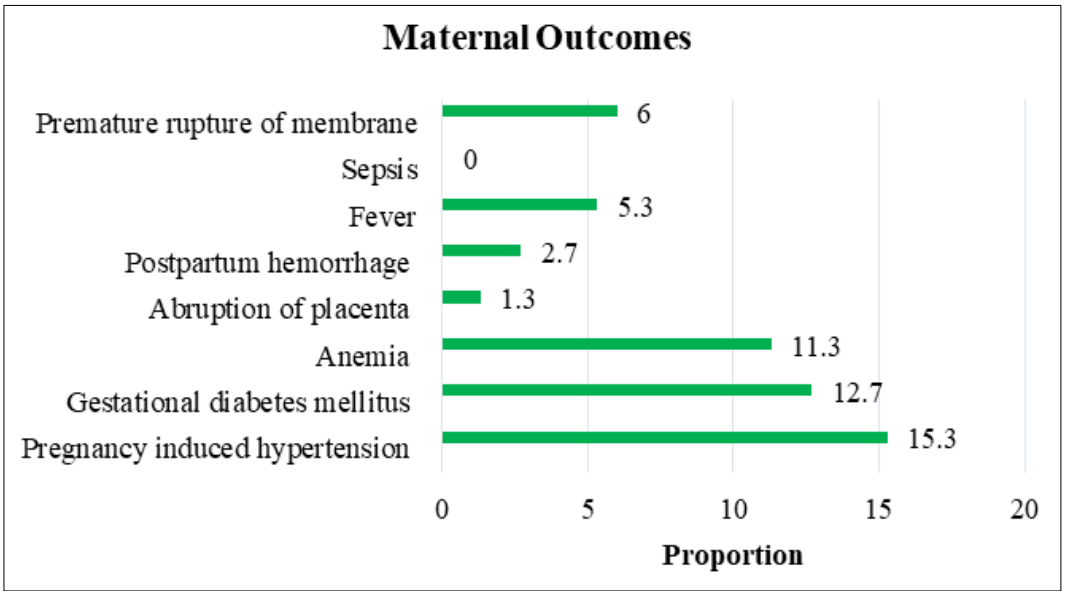
Table 3: Associations between placental characteristics and fetal outcomes

Placenta characteristics	Fetal outcomes	n (%)			P value*
USG placental thickness		**<35**	**35-45**	**>45**	
	Fetal growth retardation				
	Present	28(77.7)	11 (10.3)	0 (0.0)	<0.001
	NICU admission				
	Present	31 (86.1)	21(19.8)	5(62.5)	<0.001
	Preterm birth				
	Present	19 (52.7)	19 (17.9)	0 (0.0)	<0.001
	Birth weight (kg)				
	<2.5	6(16.6)	95(89.6)	8 (100.0)	<0.001
Placental weight (gm)		<400	400-500	>500	
	Fetal growth retardation				
	Present	31 (75.6)	7 (11.8)	1 (2.0)	<0.001
	NICU admission				
	Present	37(90.2)	9 (15.2)	11(22.0)	<0.01
	Preterm birth				
	Present	21 (51.2)	14 (23.7)	3(6.0)	<0.001
	Birth weight (kg)				
	<2.5	0(0.0)	59 (100.0)	50 (100.0)	<0.001
Placental thickness		<2.5	2.5-4.0	>4.0	
	Fetal growth retardation				
	Present	2 (100.0)	24(50.0)	13 (13.0)	<0.001
	NICU admission				
	Present	2 (100.0)	30(62.5)	25(25.0)	<0.001

	Preterm birth				
	Present	2 (100.0)	18 (37.5)	18(18.0)	0.002
	Birth weight (kg)				
	<2.5	0(0.0)	21(43.7)	88(88.0)	<0.001
Placental diameter		<18	18-21	>21	
	Fetal growth retardation				
	Present	36(50.7)	2 (4.2)	1 (3.1)	<0.001
	NICU admission				
	Present	39 (54.9)	7(14.8)	11 (34.3)	<0.001
	Preterm birth				
	Present	29(40.8)	7 (14.8)	2(6.2)	<0.001
	Birth weight (kg)				
	<2.5	30(42.2)	47 (100.0)	32 (100.0)	<0.001

Table 4: Associations between placental characteristics and maternal outcomes

Placenta characteristics	**Maternal outcomes**	**n (%)**			**P value***
Placental weight (gm)		**<400**	**400-500**	**>500**	
	Pregnancy-induced hypertension				
	Present	11(26.8)	9 (15.2)	3(6.0)	0.02
	Gestational diabetes mellitus				
	Present	1 (2.4)	2(3.3)	16 (32.0)	<0.001
	Anemia				
	Present	10 (24.3)	6 (10.1)	2 (4.0)	0.02
	Fever				
	Present	0 (0.0)	8 (13.5)	0 (0.0)	0.001
	Premature rupture of membrane				
	Present	2 (4.8)	7 (11.6)	0 (0.0)	0.03
Placental thickness		<2.5	2.5-4.0	>4.0	
	Pregnancy-induced hypertension				
	Present	2 (100.0)	13 (27.0)	8(8.0)	<0.001
	Anemia				
	Present	2 (100.0)	8 (16.6)	8 (8.0)	<0.001
Placental diameter		<18	18-21	>21	
	Pregnancy-induced hypertension				
	Present	17(23.9)	3 (6.3)	3 (9.3)	0.02
	Gestational diabetes mellitus				
	Present	1(1.4)	4(8.5)	14(43.7)	<0.001
	Anemia				
	Present	14 (19.7)	2 (4.2)	2 (6.2)	0.02



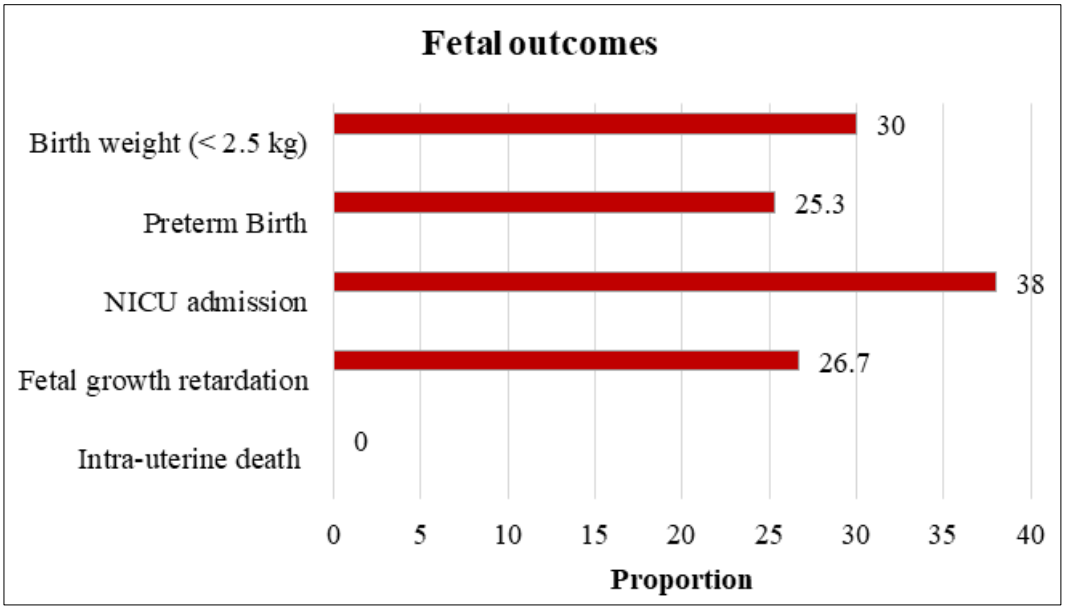


Fig 1: Adverse A) maternal and B) fetal outcomes

Discussion

The study assessed fetomaternal outcomes and placental morphology among 150 participants. In this study most placentas followed normal growth patterns, some deviations could have clinical implications, particularly regarding placental insufficiency, FGR, or maternal conditions affecting placental development [12]. Fetal Doppler studies revealed that 28.7% of participants exhibited signs of insufficiency, while 71.3% had results within normal limits. Despite some cases of Doppler abnormalities, 100% of histopathological examinations were normal, indicating no significant structural placental pathology.

The prevalence of pregnancy-induced hypertension in this study was 16%. Dhinwa *et al*. reported prevalence in India as 11% (5%-17%) [13]. In this study, 13.3% had gestational diabetes aligning with the pooled estimate of 13% (9%-16%) reported by Mantri *et al*. [14]. The prevalence of anaemia in this study was 12%, whereas NFHS-5 data reported a prevalence of 52.2% among pregnant women in India [15]. Less frequent conditions in this study included placental abruption (1.3%), postpartum haemorrhage (2.7%), fever (5.3%,), and premature rupture of membranes (6%). No cases of sepsis were reported. In line with our findings, the study by Marques *et al*. reported that placental weight, diameters, area, and BW/PW ratio were significantly lower in the hypertensive participants group [16]. Similarly, Barker *et al*. also found that hypertension prevalence decreased from 38% when the placental area was 200 cm² or less to 21% when the area was more than 320 cm² (p = 0.0007). The study concluded that the effect of placental size on hypertension is influenced by maternal nutrition, with poor maternal nutrition exacerbating the impact of smaller placental size, while better-nourished mothers may experience compensatory growth in placental surface area [17].

Other studies have also explored and found similar results, like a study by Wallace *et al*. reported that placental weight in the lower tertile (mean 484 g) and reduced placental efficiency were linked to higher risks of pre-eclampsia [18]. However, a study by Sun *et al*. found that increased placenta thickness is associated with many maternal abnormalities, including preeclampsia [12]. This could be due to differences in study populations, measurement methods, or definitions of placental thickness.

We also observed significant associations between anemia and these placental characteristics. It was more common in the <400 g placental weight group (23.3% vs. >500 g: 4.8%), in those with a placental diameter <18 cm (18.7% vs. >21 cm: 8.3%), in the <2.5 cm placental thickness group (100% vs. >4.0 cm: 8.2%), and in participants with ultrasound placental thickness <35 mm (26.3%), whereas no cases were observed in the >45 mm group. However, Mitsuda *et al*. found that PW was higher in anemic women than in those with normal haemoglobin [19]. Another study reported that higher PW is associated with anaemia [20]. Anemia was linked to smaller placental size, indicating possible placental insufficiency that could affect fetal nutrient and oxygen supply, increasing the risk of fetal growth restriction. Gestational diabetes mellitus was significantly associated only with ultrasound placental thickness. A review article reported that placentas in pregnancies with GDM are usually larger than those in pregnancies without GDM, with greater weight, volume, thickness, and diameter. However, there are some exceptions; in some cases, the placenta may be smaller than expected (below the 10th percentile in weight) [21]. However, findings across studies differ, likely due to variations in population characteristics, measurement methods, and definitions.

In this study, FGR was observed in 26.7%, NICU admission was required for 38%, Preterm birth occurred in 25.3%, and regarding birth weight, 30% had a birth weight of less than 2.5 kg. We found birth weight <2.5 was association with LBW, reduced PT, smaller diameter and lower ultrasound-measured placental thickness. These findings are in line with the literature. A study by Nascente *et al*. showed that placental weight alone explained 48% of birth weight variability [2]. Similarly, Marques *et al*. found a strong correlation between BW and PW in the presence of HDP (r = 0.79, p<0.001) [16]. In line with our study, Hutcheon *et al*. also found that high PW z scores were linked to a higher likelihood of adverse neonatal outcomes (OR 1.4), contributing to 5% of serious neonatal morbidity [22]. A study by Salafia *et al*. (2008) showed that placental weight

alone accounted for 36.6% of the variation in birth weight, while the other placental measures accounted for 28.1%. In total, all placental measures explained 39.1% of the birthweight variation [23]. A study by Haeussner *et al*. (2013) found that PW strongly correlated with BW, with the PW/BW ratio and related parameters showing consistent patterns [24]. Similar findings were reported by Grandi *et al*. [25]. Our findings align with a study by Freedman *et al*. that surface area had the strongest association with BW; a reduction in surface area correlated with a decrease in BW, even after adjusting for covariates. Reduced PT was also linked to lower BW [26]. FGR was significantly associated with lower placental weight, reduced placental thickness, smaller placental diameter and lower ultrasound-measured placental thickness in the study participants. Higher NICU admission and preterm birth were also significantly more frequent in cases with lower placental weight, thinner placentas, smaller placental diameters, and reduced ultrasound placental thickness. In line with our study, Sun *et al*. found that increased placenta thickness is associated with abnormal fetal growth [12]. Wallace *et al*. reported that placental weight in the lower tertile (mean 484 g) was linked to higher risks of preterm delivery, stillbirth, and LBW, while weight in the upper tertile (mean 788 g) was associated with increased risks of CS, post-term delivery, and high birth weight. Reduced placental efficiency was associated with higher risks of preterm delivery and LBW [18].

Conclusion

This study highlights the role of placental health in maternal and fetal outcomes. Placental weight, thickness, diameter, and ultrasound-measured thickness were closely linked to maternal conditions like pregnancy-induced hypertension and anemia, as well as fetal growth restriction, LBW, and preterm birth. While the mean placental weight (446.1 grams), thickness (4.3 cm), and diameter (17.8 cm) were within normal ranges, some cases showed reduced placental size, suggesting insufficiency that could affect fetal growth and development. Placental variations influenced maternal conditions, with smaller placental dimensions associated with a higher prevalence of PIH (16%) and anemia (12%), suggesting impaired placental function may contribute to these complications. Fetal outcomes, including fetal growth retardation (26.7%), NICU admissions (38%), and preterm birth (25.3%), were significantly linked to lower placental weight, reduced thickness, and smaller diameter. The mean birth weight (2690.5 grams) and birth length (46.4 cm) were at the lower end of the normal range, indicating a risk of IUGR in some infants.

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