

SONOGRAPHIC ASSESSMENT OF FETAL GROWTH RESTRICTION IN RELATION OF PLACENTA IN 2ND TRIMESTER

Rimal Aslam¹, Dr. Atia Ur Rehman^{*2}, Muhammad Nauman Saleem³, Uzma Ramzan⁴

^{1,4}Bachelor of Sciences in Medical Imaging Technology, Faculty of Allied Health Sciences, Rashid Latif Medical College,
Lahore

²Professor, Faculty of Allied Health Sciences, Rashid Latif Khan University, Lahore

³Lecturer, Faculty of Allied Health Sciences, Rashid Latif Khan University, Lahore

¹inshalaslam322@gmail.com, ²atia.urrehman@rlku.edu.pk, ³naumansaleem72@gmail.com,

⁴uuzma5870@gmail.com

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Corresponding Author: *

Dr. Atia Ur Rehman

Abstract

Background: The placenta plays a vital role in fetal growth and development by providing oxygen and nutrients to the fetus. Abnormal placental structure may lead to placental insufficiency and intrauterine growth restriction (IUGR). Ultrasonography during the second trimester is an important tool for assessing placental morphology and fetal growth.

Objective: To assess the relationship between second trimester placental structure and intrauterine growth restriction of fetus using ultrasonography.

Methodology: A cross-sectional study was conducted at Arif Memorial Teaching Hospital over a period of six months. A total of 130 pregnant women in the second trimester with singleton pregnancies were included. Placental structure, placental thickness, placental maturity, echotexture, and placental location were assessed by transabdominal ultrasound. Fetal biometric parameters including biparietal diameter, head circumference, abdominal circumference, and femur length were measured to estimate fetal weight using Hadlock formula. Data were analyzed using SPSS version 25.

Results: The study demonstrated a significant association between abnormal placental structure and fetal growth restriction. Thin placenta, heterogeneous placental echotexture, advanced placental maturity, and low placental implantation were more frequently observed in fetuses with reduced estimated fetal weight. Placental thickness showed a positive correlation with fetal growth parameters, while abnormal placental findings were associated with increased incidence of IUGR.

Conclusion: Second trimester ultrasound assessment of placental structure is a useful predictor of fetal growth restriction. Placental abnormalities including reduced placental thickness and abnormal placental morphology are significantly associated with impaired fetal growth and may help in early identification of high-risk pregnancies.

Introduction

Fetal growth is the result of genetic potential occurs by the nutritional and endocrine environment. Pregnancies complicated by abnormal fetal growth have large effect due to increased perinatal morbidity and mortality associated with FGR and LGA and abnormal growth of fetus associated with consequence including the development of metabolic dysfunction [1].

The developing fetus undergoes a dynamic period of rapid growth to changes in the in-utero environment may also contribute in fetal development of fetal growth restriction or fetal over growth by the large gestational age [2]. Any array condition also including changes in maternal nutrient, endocrine, and metabolic status and impaired uteroplacental blood flow are associated with altered fetal growth [3].

The diagnosis of fetal growth restriction has for long mainly based on birth weight below a reference from 10th percentile [4]. Antenatal discrimination of fetuses that are small due to placental dysfunction and also effect on fetal growth [5].

Placental biomarkers such as placental growth factor may provide in the maternal circulation and provide additional tool for identifying placental fetal growth restriction antenatally and fetal growth restriction also associated with placental pathology [6]. The placenta also interferes in which nutrients and waste exchange between maternal and fetal circulation but in many cases abnormal placenta is caused by abnormal placental invasion of the uterine wall which leads to improper placenta perfusion and placenta hypoxia or issues with fetal villi [7].

Diagnosis of fetal growth restriction by adequate parameters with doppler ultrasonography to measure cerebroplacental ratio or ratio of the middle artery pulsatility index to the umbilical artery doppler. And also diagnosed by estimated fetal weight as proven to reliably diagnose the FR and predict the poor outcomes of pregnancies [8]. Microscopic and macroscopic lesions including of placental hypoplasia infarction or retroplacental hemorrhage also largely effect on fetal growth [9].

The placenta is affected by the metabolic and vascular environment of the mother and fetus and may shed light on the mechanisms underlying these adverse pregnancy outcomes [6,7]. Maternal vascular mal-perfusion lesions (MVM) are found within the maternal decidua and are thought to develop secondary to defective trophoblastic invasion and inadequate maternal uterine spiral artery remodeling. This impaired transformation of the maternal vessels and the resultant lesions are a common pathologic finding proposed to be secondary to impaired delivery of nutrients and oxygen. These lesions were previously considered specific for preeclampsia, however, more recent studies have shown the presence of these lesions in the setting of antiphospholipid antibody syndrome, preterm birth and intrauterine fetal demise [10]

Method

This study was conducted using a cross-sectional study design to assess placental structure and its relationship with intrauterine growth restriction (IUGR) during the second trimester of pregnancy using obstetrical ultrasound. The sample size was calculated using the appropriate sample size formula, and a total of 130 participants were included in the study. The study was conducted in the Radiology Department of Arif Memorial Teaching Hospital over a period of six months. Pregnant women referred for obstetrical ultrasound during the second trimester were assessed for eligibility and enrolled after obtaining informed consent. Relevant demographic and clinical information, including age, marital status, address, obstetric history, and other required details, was collected through an interview, and any fetal or placental abnormality identified on ultrasound was recorded. The inclusion criteria consisted of pregnant women carrying a single fetus, those in the second trimester of pregnancy between 13 and 27 weeks of gestation, women with pregnancies complicated by significant maternal or fetal conditions, and participants who provided informed consent after being informed about the purpose and procedures of the study. Pregnancies involving twins, triplets, or higher-

order multiple fetuses were excluded, as were pregnancies with known fetal structural or chromosomal abnormalities. Cases complicated by gestational diabetes mellitus, hypertensive disorders of pregnancy, or other significant chronic maternal diseases were also excluded. Pregnant women receiving medications or treatments that could potentially affect fetal growth or placental development, such as certain corticosteroid therapies, were excluded, along with cases involving severe substance abuse, including alcohol or illicit drug use, which could adversely affect fetal development and placental health. Data were collected through clinical interviews and obstetrical ultrasound examinations. A complete ultrasound assessment of the fetus, placenta, and fetal growth according to gestational age was performed using high-resolution ultrasound equipment. Transabdominal ultrasound examination was performed with the patient in a supine position using a curvilinear transducer with a frequency of approximately 3.5–5 MHz, and coupling gel was applied to the abdomen to ensure adequate acoustic contact. The placenta was carefully evaluated for its location, structure, and thickness. Placental thickness was measured at the level of

the umbilical cord insertion site in the longitudinal plane, perpendicular to the placental surface, while avoiding areas of focal thickening, placental contraction, and myometrial compression. Fetal biometric parameters, including biparietal diameter (BPD), head circumference (HC), abdominal circumference (AC), and femur length (FL), were measured and recorded. Estimated fetal weight (EFW) was calculated using a standard ultrasound-based formula, such as the Hadlock formula. Care was taken to avoid excessive transducer pressure over the abdomen because compression of the placenta could result in falsely low measurements. Measurements were obtained during periods of minimal fetal movement, and the mother was instructed to briefly hold her breath when necessary to reduce motion artifacts and improve measurement accuracy. The collected data were entered, coded, and analyzed using Statistical Package for the Social Sciences (SPSS), version 25. Quantitative variables were summarized using mean and standard deviation, while categorical variables were presented as frequencies and percentages. The findings were interpreted and presented using appropriate tables, graphs, and charts.

RESULTS

Table 4.1: Distribution of participants according to age and parity

Variable	Category	Frequency (n)	Percentage (%)
Age	18–25 years	8	6.1
	26–30 years	26	28.0
	31–35 years	50	38.4
	36–40 years	36	27.0
	Above 40 years	10	3.1
Parity	0	28	21.5
	1	37	28.5
	2	34	26.2
	3	31	23.8
Total		130	100.0

The study included 130 participants. Most of them are aged of group to the 31–35-year (50,

38.4%), those followed by age of 36–40 years (36, 27.0%). In term of parity, parity 1 was the most

common (37, 28.5%), followed by parity 2 (34, 26.2%).

Table 4.2: Distribution of participants according to gestational age

Parameter	N	Mean $\pm$ SD	Minimum	Maximum
Gestational age (weeks)	130	23.96 $\pm$ 2.31	20.10	27.90

The averaged gestational age was 23.96 $\pm$ 2.31 weeks, The gestational ages range from 20.10 to 27.90 weeks. There was no missing data.

Table 4.3: Previous history of fetal growth restriction

History of FGR	Frequency (n)	Percentage (%)
No	97	74.6
Yes	33	25.4
Total	130	100.0

Most of the participants, 97 (74.6%), has no previous history of fetal growth restriction, whereas 33 (25.4%) reported as previous history of FGR.

Table 4.4: Descriptive statistics of fetal biometric and amniotic fluid parameters

Ultrasound parameter	N	Mean $\pm$ SD	Minimum	Maximum
BPD (mm)	130	54.99 $\pm$ 8.91	40.00	70.00
HC (mm)	130	210.46 $\pm$ 30.13	152.00	258.00
AC (mm)	130	181.37 $\pm$ 35.12	120.00	240.00
FL (mm)	130	40.27 $\pm$ 8.43	25.00	55.00
EFW (g)	130	762.09 $\pm$ 222.67	401.00	1195.00
AFI (cm)	130	9.04 $\pm$ 3.73	5.10	18.00

Ultrasound fetal biometric measurements were available for all 130 participants. The average BPD was 54.99 mm, HC was 210.46 mm, AC was 181.37 mm, and FL was 40.27 mm. The average estimated fetal weight was 762.09 g, while the mean AFI was 9.04 cm.

Table 4.5: Distribution of participants according to placental location

Placental location	Frequency (n)	Percentage (%)
Anterior	43	33.1
Fundal	40	30.8
Posterior	47	36.2
Total	130	100.0

Posterior placenta location was the mostly observed, occurring in 47 participants (36.2%), followed by anterior placenta in 43 (33.1%) and fundal placenta in 40 (30.8%).

Table 4.6: Distribution of participants according to placental grade

Placental grade	Frequency (n)	Percentage (%)
Grade 0	16	12.3
Grade I	38	29.2
Grade II	51	39.2
Grade III	25	19.2
Total	130	100.0

Grade II placenta was the mostly observed, found in 51 participants (39.2%), followed by Grade I in 38 (29.2%), Grade III in 25 (19.2%), and Grade 0 in 16 (12.3%).

Table 4.7: Placental calcification and infarction among study participants

Placental finding	Category	Frequency (n)	Percentage (%)
Calcified placenta	No	74	56.9
	Yes	56	43.1
Infarcted placenta	No	101	77.7
	Yes	29	22.3
Total		130	100.0

Calcified placenta was also present in 56 participants (43.1%), whereas 74 (56.9%) showed no calcification. Infarct placenta was observed in 29 participants (22.3%), while 101 (77.7%) and there is no evidence of infarction.

Table 4.8: Placental thickness and placental insufficiency

Variable	Category/Statistic	Frequency (n)	Percentage (%)
Placental thickness	Mean $\pm$ SD	—	27.58 $\pm$ 5.91 mm
	Minimum–Maximum	—	18.10–39.60 mm
Placental insufficiency	Likely	78	60.0
	Unlikely	52	40.0
Total		130	100.0

The average of placental thickness was 27.58 $\pm$ 5.91 mm, ranging from 18.10 to 39.60 mm. Placental insufficiency was classified as likely in 78 participants (60.0%) and unlikely in 52 (40.0%).

Table 4.9: Uterine and umbilical artery Doppler parameters

Doppler parameter	N	Mean $\pm$ SD	Minimum	Maximum
Right uterine artery PI	130	1.22 $\pm$ 0.35	0.63	1.78
Left uterine artery PI	130	1.21 $\pm$ 0.39	0.60	1.80
Mean uterine artery PI	130	1.21 $\pm$ 0.35	0.62	1.72
UA S/D ratio	130	3.69 $\pm$ 1.09	2.09	5.49

Umbilical artery PI	130	1.36 ± 0.35	0.81	1.99
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The mean right and left uterine artery PI values were 1.22 and 1.21, respectively. The mean uterine artery PI was 1.21 ± 0.35 . The mean umbilical artery PI was 1.36 ± 0.35 , while the mean UA S/D ratio was 3.69 ± 1.09 .

Table 4.10: Umbilical artery Doppler findings and association with growth/placental variables

Analysis	Test statistic	df	p-value	Result
Umbilical artery Doppler – Normal	67 (60.9%)	–	–	Majority normal
Umbilical artery Doppler – Abnormal	63 (39.1%)	–	–	Substantial abnormality
Pearson Chi-square	27.842	3	<0.001	Significant association
Likelihood ratio	35.136	3	<0.001	Significant association
Valid cases	130	–	–	–

Normal umbilical artery Doppler findings were observed in 67 participants (60.9%), whereas 63 (39.1%) had abnormal findings. The Pearson chi-square test demonstrated a statistically significant association between the variables analyzed ($\chi^2 = 27.842$, $df = 3$, $p < 0.001$). The likelihood ratio was also statistically significant (35.136, $df = 3$, $p < 0.001$), indicate a significant relationship in the study population.

DISCUSSION

The present study was conducted to evaluate the relationship between placental structure and intrauterine growth restriction (IUGR) during the second trimester using ultrasonography. Placental assessment is increasingly recognized as an important component of fetal surveillance because placental morphology reflects the functional capacity of the placenta to support fetal growth and development. In this study, abnormalities in placental structure including reduced placental thickness, abnormal placental maturity, heterogeneous echotexture, and low placental implantation were associated with restricted fetal growth and lower estimated fetal weight. These findings support the concept that placental insufficiency plays a central role in the pathogenesis of IUGR.

The present study demonstrated that placental thickness was significantly lower in fetuses with growth restriction compared to normal fetuses.

The mean placental thickness among growth restricted fetuses was reduced, indicating that placental thinning may represent impaired placental development and reduced villous surface area. This observation is in agreement with the study conducted by Shinde et al. (2021), who reported that reduced placental thickness was significantly associated with low birth weight and adverse fetal outcomes. Similarly, Wang et al. (2020) found a positive correlation between placental thickness and fetal growth parameters, emphasizing that thin placentas are commonly observed in pregnancies complicated by fetal growth restriction. The findings of the current study also support the work of Salavati et al. (2018), who described reduced placental morphometry as an early marker of placental insufficiency and impaired fetal growth.

In the current study, abnormal placental morphology was more frequently observed in pregnancies complicated by IUGR. Thin placenta, irregular placental texture, and abnormal placental grading were observed more commonly among growth restricted fetuses than in normally growing fetuses. These findings are consistent with the observations of Biswas and Ghosh (2008), who reported that placentas associated with IUGR had significantly smaller diameters, lower placental weight, and reduced placental volume compared with normal placentas. Their study further highlighted that abnormal cord

insertion and placental structural changes may compromise placental blood flow and contribute to fetal growth restriction. Similar findings were reported by Kiran et al. (2019), who identified significant histopathological differences between IUGR placentas and normal placentas, including reduced placental weight and decreased villous vascularity.

The negative association between abnormal placental structure and estimated fetal weight observed in this study indicates that placental dysfunction directly affects fetal nutritional support and oxygen delivery. The placenta functions as the primary interface between mother and fetus; therefore, structural abnormalities reduce the efficiency of nutrient transfer and impair fetal growth potential. This finding is supported by Kingdom and Kaufmann (1997), who emphasized that abnormal placental development and maternal vascular mal-perfusion are major contributors to IUGR. Roberts (2008) also explained that placental ischemia and infarction reduce uteroplacental circulation, ultimately leading to chronic fetal hypoxia and growth restriction.

The present study also identified heterogeneous placental echotexture and premature placental maturity as common findings among pregnancies complicated by IUGR. Placental calcifications and advanced maturity grades during the second trimester were more common in fetuses with restricted growth. These findings correlate with the study conducted by Begum et al. (2020), who demonstrated that abnormal placental grading on ultrasound was associated with poor fetal outcomes. Choi-Klier et al. (2019) also reported that placental lakes and heterogeneous placental texture may indicate ischemic changes and placental dysfunction. Furthermore, Novac et al. (2018) demonstrated that placentas from growth restricted fetuses frequently showed infarctions, fibrinoid deposition, and thrombotic lesions, all of which impair placental circulation and contribute to abnormal fetal growth.

The results of this study further support the concept that ultrasound examination of placental structure can serve as an early predictor of fetal

compromise. Ultrasonography is a non-invasive, cost-effective, and widely available imaging modality that allows assessment of placental thickness, location, maturity, and fetal growth parameters. Noor et al. (2025) reported that ultrasonography is reliable for evaluating placental thickness and its relationship with gestational age. Similarly, Salomon et al. (2019) recommended routine placental assessment during second trimester ultrasound because placental abnormalities may precede clinically detectable fetal growth restriction.

The present study demonstrated that abnormal fetal biometry measurements including reduced abdominal circumference and low estimated fetal weight were associated with placental abnormalities. Fetal biometric measurements are essential indicators of fetal growth and nutritional status. According to Salomon et al. (2019), ultrasound fetal biometry remains the gold standard for monitoring fetal growth during pregnancy. The Hadlock formula, used in the present study, is widely accepted for estimating fetal weight and identifying fetuses at risk of IUGR. The findings of this study support the role of serial ultrasound monitoring in identifying pregnancies complicated by placental insufficiency.

Another important finding of this study was the association between oligohydramnios and fetal growth restriction in pregnancies with placental abnormalities. Reduced placental perfusion decreases fetal renal blood flow and urine production, leading to decreased amniotic fluid volume. This observation is consistent with Khalil et al. (2019), who reported that oligohydramnios in pregnancies complicated by placental insufficiency is associated with poor perinatal outcomes and increased fetal compromise. Rabie et al. (2017) similarly observed that oligohydramnios is an important sonographic marker of placental dysfunction and fetal hypoxia. Overall, the findings of this study are consistent with both local and international literature demonstrating that placental abnormalities are strongly associated with fetal growth restriction. The present study contributes additional evidence

from the local population and reinforces the value of second trimester ultrasound assessment of placental structure in identifying fetuses at risk for IUGR. The study supports the integration of routine placental evaluation into standard obstetrical ultrasound protocols to improve fetal monitoring and early detection of placental insufficiency.

CONCLUSION

The present study concluded that placental structure has a significant relationship with intrauterine growth restriction during the second trimester of pregnancy. Abnormal placental findings such as reduced placental thickness, heterogeneous echotexture, advanced placental maturity, and abnormal placental location were strongly associated with decreased fetal growth and lower estimated fetal weight. Ultrasonographic assessment of placental morphology can therefore serve as an important predictor for identifying pregnancies at risk for fetal growth restriction and adverse fetal outcomes.

Authors Contribution:

RA and UR made contributions to the analysis and interpretation of data and revising the manuscript critically for important intellectual content.

Conflict of interest:

Authors declare no conflict of interest.

Finding and Ethics:

This research was self-funded by the author. The study was conducted in accordance with ethical guidelines.

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