Section: Radiology

Original Research Article

ROLE OF FETAL PANCREATIC BIOMETRY IN PREDICTING GESTATIONAL DIABETES MELLITUS AND FETAL GROWTH RESTRICTION: A PROSPECTIVE OBSERVATIONAL STUDY

Nilofar Khudbuddin Mujawar[1], Pravin R. Bhosale[2]

[1]Assistant Professor, Department of Radio-Diagnosis, Prakash Institute of Medical Science and Research, Urun-Ishwarpur, Maharashtra, India
[2]Associate Professor, Department of Radiology, Government Medical College and Hospital, Miraj, Maharashtra, India

Received *: 17/04/2026*
Received in revised form *: 04/06/2026*
Accepted *: 20/06/2026*

Corresponding Author:
Dr. Nilofar Khudbuddin Mujawar, Assistant Professor, Department oF Radio-diagnosis, Prakash Institute of Medical Science and Research, Urun-Ishwarpur, Maharashtra, India.
Email: dr.nilumujawar@gmail.com

DOI: 10.70034/ijmedph.2026.3.90

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (3); 550-555

ABSTRACT

Background: Gestational diabetes mellitus (GDM) and fetal growth restriction (FGR) are major causes of maternal and perinatal morbidity. Early identification of pregnancies at risk remains challenging. Fetal pancreatic biometry has emerged as a potential ultrasonographic marker reflecting fetal metabolic status and growth, with possible utility in predicting both GDM and FGR. The aim is to evaluate the role of fetal pancreatic biometry in predicting gestational diabetes mellitus and fetal growth restriction during pregnancy.

Materials and Methods: This prospective observational study included 80 pregnant women with singleton pregnancies attending a tertiary care teaching hospital. Fetal pancreatic dimensions, including transverse diameter, thickness, and circumference, were measured using obstetric ultrasonography between 22–24 weeks and 28–30 weeks of gestation. Participants underwent routine screening for GDM using oral glucose tolerance testing and were followed until delivery. Pregnancy outcomes, fetal growth parameters, birth weight, and neonatal outcomes were recorded. Statistical analysis was performed using appropriate parametric and non-parametric tests, and a p-value <0.05 was considered statistically significant

Results: The mean maternal age was 27.3 ± 4.6 years, and GDM was diagnosed in 23.8% of participants, while FGR occurred in 13.8%. Fetal pancreatic dimensions increased significantly with gestational age (p<0.001). Pregnancies complicated by GDM demonstrated significantly larger pancreatic transverse diameter (20.3 ± 2.4 mm vs. 17.1 ± 2.1 mm), thickness (8.7 ± 1.2 mm vs. 7.0 ± 1.1 mm), and circumference (56.4 ± 6.1 mm vs. 47.7 ± 5.8 mm) compared with non-GDM pregnancies (p<0.001). Conversely, pregnancies with FGR or adverse fetal outcomes showed significantly smaller pancreatic dimensions, including circumference (42.6 ± 5.3 mm vs. 51.9 ± 6.2 mm; p<0.001). Reduced pancreatic biometry was significantly associated with low birth weight and NICU admission

Conclusion: Fetal pancreatic biometry is significantly associated with both gestational diabetes mellitus and fetal growth restriction. Increased pancreatic dimensions correlate with GDM, whereas reduced pancreatic measurements are associated with FGR and adverse neonatal outcomes. Fetal pancreatic biometry may serve as a valuable adjunctive sonographic marker for identifying pregnancies at risk for metabolic and growth-related complications.

Keywords: Fetal Pancreatic Biometry. Gestational Diabetes Mellitus. Fetal Growth Restriction (FGR).

INTRODUCTION

Gestational diabetes mellitus (GDM) and fetal growth restriction (FGR) are among the most common and clinically significant complications encountered during pregnancy. GDM is defined as glucose intolerance with onset or first recognition during pregnancy and is associated with increased maternal and fetal morbidity, including macrosomia, neonatal hypoglycemia, birth trauma, and future metabolic disorders. Conversely, FGR refers to the failure of a fetus to achieve its genetically determined growth potential and is associated with increased risks of perinatal mortality, neonatal complications, and long-term neurodevelopmental impairment. Early identification of pregnancies at risk for GDM and FGR remains a major challenge in obstetric practice and is essential for timely intervention and improved perinatal outcomes.[1]

Ultrasonography has become an indispensable tool for fetal assessment and monitoring during pregnancy. Conventional fetal biometric parameters such as biparietal diameter, head circumference, abdominal circumference, and femur length are routinely used to assess fetal growth. However, recent advances in fetal imaging have led to increased interest in the evaluation of fetal organ biometry as potential markers of intrauterine metabolic and growth disturbances. Among these, fetal pancreatic biometry has emerged as a promising parameter due to the critical role of the pancreas in fetal glucose metabolism and growth regulation.[2]

The fetal pancreas responds to maternal glucose levels through alterations in insulin secretion. Maternal hyperglycemia results in fetal hyperinsulinemia, which may stimulate pancreatic hypertrophy and enlargement. Studies have demonstrated that fetuses of mothers with GDM often exhibit increased pancreatic dimensions compared to those of normoglycemic mothers. Consequently, measurement of fetal pancreatic size may serve as an early sonographic marker for the prediction and monitoring of GDM. Furthermore, pancreatic growth is closely related to fetal nutritional status and insulin-mediated growth processes, suggesting its potential utility in assessing abnormal fetal growth patterns.[3]

Fetal growth restriction is characterized by impaired nutrient and oxygen supply to the fetus, leading to alterations in organ development and growth. Reduced pancreatic size has been reported in growth-restricted fetuses, reflecting compromised endocrine and exocrine pancreatic development. Therefore, fetal pancreatic biometry may provide additional information regarding fetal growth and metabolic status beyond conventional biometric measurements. Despite encouraging findings, the clinical utility of fetal pancreatic measurements in predicting GDM and FGR remains inadequately explored, particularly in the Indian population.[4]

Aim: To evaluate the role of fetal pancreatic biometry in predicting gestational diabetes mellitus and fetal growth restriction during pregnancy.

Objectives

1. To measure fetal pancreatic dimensions using obstetric ultrasonography at predefined gestational ages.
2. To assess the association between fetal pancreatic biometry and the development of gestational diabetes mellitus.
3. To determine the predictive value of fetal pancreatic biometry for fetal growth restriction and adverse fetal outcomes.

MATERIALS AND METHODS

The data were collected from pregnant women attending the Antenatal Outpatient Department and admitted to the Department of Obstetrics and Gynecology of a tertiary care teaching hospital. Clinical, laboratory, ultrasonographic, and pregnancy outcome data were obtained from enrolled participants.

Study Design: Prospective observational study.

Study Location: The study was conducted in the Department of Obstetrics and Gynecology in collaboration with the Department of Radiodiagnosis at a tertiary care teaching hospital.

Study Duration: The study was conducted over a period of 18 months, including patient recruitment, follow-up, data collection, analysis, and report preparation.

Sample Size: A total of 80 pregnant women fulfilling the eligibility criteria were included in the study.

Inclusion Criteria

- Pregnant women with singleton pregnancy.
- Gestational age between 20 and 24 weeks confirmed by first-trimester ultrasound or reliable last menstrual period.
- Women willing to provide written informed consent.
- Women available for follow-up until delivery.

Exclusion Criteria

- Pre-existing diabetes mellitus (Type 1 or Type 2).
- Multiple pregnancies.
- Major fetal congenital anomalies detected on ultrasonography.
- Maternal chronic medical disorders affecting fetal growth (chronic hypertension, renal disease, autoimmune disorders).
- Pregnancies complicated by chromosomal abnormalities.
- Inadequate visualization of the fetal pancreas during ultrasonographic examination.

Procedure and Methodology: After obtaining approval from the Institutional Ethics Committee and written informed consent, eligible pregnant women were enrolled consecutively. Detailed maternal history including age, parity, body mass index, obstetric history, and family history of diabetes was recorded.

All participants underwent routine obstetric ultrasonography between 20 and 24 weeks of gestation. Fetal pancreatic biometry was measured using a high-resolution ultrasound machine with a curvilinear transducer. The pancreas was identified in the transverse section of the fetal abdomen between the stomach and spine. Pancreatic circumference, transverse diameter, and thickness were measured according to standardized protocols. Three measurements were obtained and the average value was considered for analysis.

Routine fetal biometric parameters including biparietal diameter, head circumference, abdominal circumference, and femur length were also recorded. Participants subsequently underwent a 75-g oral glucose tolerance test (OGTT) between 24 and 28 weeks of gestation. Diagnosis of GDM was made according to standard international criteria.

Patients were followed throughout pregnancy with regular antenatal visits. Serial growth assessments and Doppler studies were performed whenever clinically indicated. Fetal growth restriction was diagnosed based on estimated fetal weight below the 10th percentile for gestational age with supportive Doppler findings.

Maternal and neonatal outcomes including gestational age at delivery, birth weight, neonatal complications, and mode of delivery were documented.

Sample Processing: Ultrasonographic measurements were recorded on structured case record forms immediately after examination. Laboratory reports including OGTT results were verified and entered into the study database. All data were checked for completeness and accuracy before statistical analysis.

Statistical Methods: Data were entered into Microsoft Excel and analyzed using Statistical Package for Social Sciences (SPSS) version 25.0.

- Continuous variables were expressed as mean ± standard deviation (SD).
- Categorical variables were expressed as frequency and percentage.
- Student's t-test or Mann–Whitney U test was used for comparison of continuous variables.
- Chi-square test or Fisher's exact test was used for categorical variables.
- Pearson or Spearman correlation coefficients were calculated to assess associations between pancreatic measurements and fetal growth parameters.
- Receiver Operating Characteristic (ROC) curve analysis was performed to determine the predictive accuracy and optimal cut-off values of fetal pancreatic biometry for GDM and FGR.
- Multivariate logistic regression analysis was performed to identify independent predictors of GDM and FGR.
- A p-value <0.05 was considered statistically significant.

Data Collection: A predesigned and pretested case record form was used for data collection. The following information was collected:

- Maternal demographic details and obstetric history.
- Maternal BMI and antenatal risk factors.
- Fetal pancreatic measurements (diameter, thickness, circumference).
- Routine fetal biometric parameters.
- OGTT results and diagnosis of GDM.
- Serial fetal growth assessments.
- Diagnosis of fetal growth restriction.
- Doppler findings when applicable.
- Gestational age at delivery.
- Birth weight and neonatal outcomes.
- Maternal and fetal complications during pregnancy and delivery.

RESULTS

Table 1. Baseline Profile and Pregnancy Outcomes of Study Participants (N=80)

Variable	Value n (%) / Mean ± SD	Test value	95% CI	p-value
Maternal age (years)	27.3 ± 4.6	t=52.99	26.3–28.3	<0.001*
Gestational age at enrolment (weeks)	22.7 ± 1.3	t=156.19	22.4–23.0	<0.001*
BMI (kg/m²)	24.8 ± 3.7	t=59.96	24.0–25.6	<0.001*
Primigravida	43 (53.8)	χ^2=0.45	42.9–64.3	0.502
Multigravida	37 (46.2)		35.7–57.1	
GDM diagnosed	19 (23.8)	χ^2=22.05	15.8–34.1	<0.001*
FGR diagnosed	11 (13.8)	χ^2=42.05	7.8–22.9	<0.001*
Normal fetal outcome	62 (77.5)	χ^2=24.20	67.2–85.3	<0.001*
Adverse fetal outcome	18 (22.5)		14.7–32.8	

[Table 1] presents the baseline characteristics and pregnancy outcomes of the 80 study participants. The mean maternal age was 27.3 ± 4.6 years (95% CI: 26.3–28.3), while the mean gestational age at enrolment was 22.7 ± 1.3 weeks (95% CI: 22.4–23.0). The average maternal BMI was 24.8 ± 3.7 kg/m² (95% CI: 24.0–25.6). Slightly more than half of the women were primigravida (53.8%), whereas 46.2% were multigravida, with no significant difference in gravidity distribution (p=0.502). Gestational diabetes mellitus (GDM) was diagnosed in 19 women (23.8%), and fetal growth restriction (FGR) was identified in 11 pregnancies (13.8%). Overall, normal fetal outcomes were observed in 62 cases (77.5%), while adverse fetal outcomes occurred in 18 cases (22.5%).

Table 2. Fetal Pancreatic Dimensions at Predefined Gestational Ages (N=80)

Variable	Value Mean ± SD	Test value	95% CI	p-value
Pancreatic transverse diameter at 22–24 weeks (mm)	13.7 ± 2.1	t=58.37	13.2–14.2	<0.001*
Pancreatic thickness at 22–24 weeks (mm)	5.8 ± 1.1	t=47.17	5.6–6.0	<0.001*
Pancreatic circumference at 22–24 weeks (mm)	38.6 ± 5.4	t=63.95	37.4–39.8	<0.001*
Pancreatic transverse diameter at 28–30 weeks (mm)	17.9 ± 2.6	t=61.56	17.3–18.5	<0.001*
Pancreatic thickness at 28–30 weeks (mm)	7.4 ± 1.3	t=50.91	7.1–7.7	<0.001*
Pancreatic circumference at 28–30 weeks (mm)	49.8 ± 6.7	t=66.47	48.3–51.3	<0.001*
Increase in pancreatic circumference	11.2 ± 3.8	t=26.36	10.4–12.0	<0.001*

[Table 2] summarizes fetal pancreatic measurements obtained at different gestational ages. At 22–24 weeks of gestation, the mean pancreatic transverse diameter was 13.7 ± 2.1 mm, pancreatic thickness was 5.8 ± 1.1 mm, and pancreatic circumference was 38.6 ± 5.4 mm. By 28–30 weeks, these dimensions increased significantly, with mean transverse diameter reaching 17.9 ± 2.6 mm, thickness 7.4 ± 1.3 mm, and circumference 49.8 ± 6.7 mm. The mean increase in pancreatic circumference during the observation period was 11.2 ± 3.8 mm (95% CI: 10.4–12.0). All pancreatic biometric parameters demonstrated highly significant growth with advancing gestation (p<0.001)

Table 3. Association Between Fetal Pancreatic Biometry and GDM (N=80)

Variable	GDM Present (n=19)	GDM Absent (n=61)	Test value	95% CI	p-value
Pancreatic transverse diameter at 28–30 weeks (mm)	20.3 ± 2.4	17.1 ± 2.1	t=5.59	2.1–4.3	<0.001*
Pancreatic thickness at 28–30 weeks (mm)	8.7 ± 1.2	7.0 ± 1.1	t=5.75	1.1–2.3	<0.001*
Pancreatic circumference at 28–30 weeks (mm)	56.4 ± 6.1	47.7 ± 5.8	t=5.66	5.6–11.8	<0.001*
Increased pancreatic biometry	15 (78.9)	18 (29.5)	χ^2=14.78	28.1–68.7	<0.001*
Normal pancreatic biometry	4 (21.1)	43 (70.5)			
Mean birth weight (kg)	3.18 ± 0.42	2.82 ± 0.39	t=3.45	0.15–0.57	0.001*

[Table 3] evaluates the association between fetal pancreatic biometry and the development of gestational diabetes mellitus. Fetuses of mothers who developed GDM exhibited significantly larger pancreatic dimensions at 28–30 weeks compared to those without GDM. The mean pancreatic transverse diameter was 20.3 ± 2.4 mm in the GDM group versus 17.1 ± 2.1 mm in the non-GDM group (p<0.001). Similarly, pancreatic thickness was significantly higher among GDM pregnancies (8.7 ± 1.2 mm vs. 7.0 ± 1.1 mm, p<0.001), as was pancreatic circumference (56.4 ± 6.1 mm vs. 47.7 ± 5.8 mm, p<0.001). Increased pancreatic biometry was observed in 78.9% of GDM cases compared with only 29.5% of non-GDM pregnancies (χ^2=14.78, p<0.001). Furthermore, the mean birth weight was significantly greater among infants born to mothers with GDM (3.18 ± 0.42 kg) than those without GDM (2.82 ± 0.39 kg; p=0.001).

Table 4. Predictive Value of Fetal Pancreatic Biometry for FGR and Adverse Fetal Outcomes (N=80)

Variable	FGR / Adverse Outcome Present (n=18)	Outcome Normal (n=62)	Test value	95% CI	p-value
Pancreatic transverse diameter at 28–30 weeks (mm)	15.8 ± 2.2	18.5 ± 2.4	t=4.31	-3.9 to -1.5	<0.001*
Pancreatic thickness at 28–30 weeks (mm)	6.4 ± 1.0	7.7 ± 1.2	t=4.17	-1.9 to -0.7	<0.001*
Pancreatic circumference at 28–30 weeks (mm)	42.6 ± 5.3	51.9 ± 6.2	t=5.77	-12.5 to -6.1	<0.001*
Reduced pancreatic biometry	13 (72.2)	9 (14.5)	χ^2=22.59	36.8–74.1	<0.001*
Normal pancreatic biometry	5 (27.8)	53 (85.5)			
Low birth weight	12 (66.7)	8 (12.9)	χ^2=21.29	32.4–70.4	<0.001*
NICU admission	7 (38.9)	6 (9.7)	χ^2=8.50	10.2–49.2	0.004*

*Statistically significant at p<0.05.

[Table 4] demonstrates the predictive value of fetal pancreatic biometry for fetal growth restriction and adverse fetal outcomes. Pregnancies complicated by FGR or adverse outcomes showed significantly smaller fetal pancreatic dimensions compared with pregnancies having normal outcomes. The mean pancreatic transverse diameter was 15.8 ± 2.2 mm in the affected group compared with 18.5 ± 2.4 mm in the normal outcome group (p<0.001). Likewise, pancreatic thickness (6.4 ± 1.0 mm vs. 7.7 ± 1.2 mm) and pancreatic circumference (42.6 ± 5.3 mm vs. 51.9 ± 6.2 mm) were significantly reduced among fetuses with adverse outcomes (p<0.001 for both). Reduced pancreatic biometry was present in 72.2% of pregnancies with FGR or adverse outcomes compared with only 14.5% of normal pregnancies

(χ^2=22.59, p<0.001). Low birth weight occurred in 66.7% of affected pregnancies versus 12.9% of normal pregnancies (p<0.001), while NICU admission was required in 38.9% and 9.7% of cases, respectively (p=0.004).

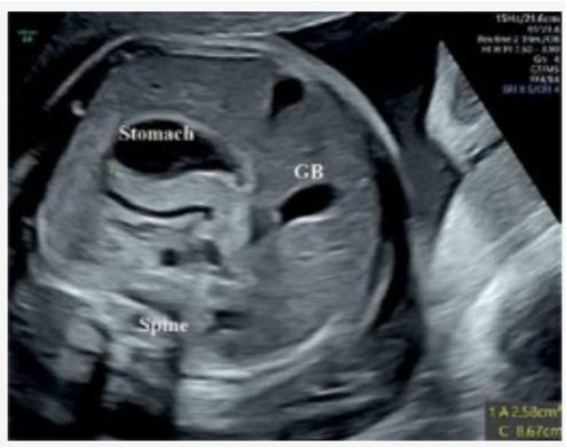

Figure 1: Fetal pancreas imaging: The best angle of imaging of the fetal pancreas is rather narrow. It consists of a transverse, antero-posterior abdominal section, at the level between the stomach and the spine-aorta- IVC complex

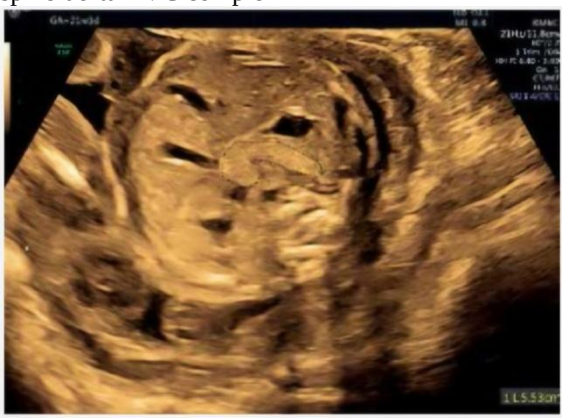

Figure 2: Image of the mildly hyperechogenic pancreas measured above 95th percentile for gestational age at 21 weeks in a patient diagnosed 4 weeks later with GDM

DISCUSSION

In the present study, the mean maternal age was 27.3±4.6 years, mean gestational age at enrolment was 22.7±1.3 weeks and mean BMI was 24.8±3.7 kg/m². GDM was diagnosed in 19 cases (23.8%), FGR in 11 cases (13.8%) and adverse fetal outcome in 18 cases (22.5%). These findings suggest that the study population had a meaningful burden of metabolic and growth-related pregnancy complications. Similar observations were reported by Gilboa et al. (2021),[1] who emphasized that maternal glycemic status influences fetal pancreatic size, particularly in pregnancies complicated by GDM. Chew et al. (2024),[2] also stated that FGR is associated with increased neonatal morbidity and requires careful fetal biometric and Doppler assessment.

In the present study, fetal pancreatic dimensions increased significantly with advancing gestational age. Pancreatic transverse diameter increased from 13.7±2.1 mm at 22–24 weeks to 17.9±2.6 mm at 28–30 weeks, thickness increased from 5.8±1.1 mm to 7.4±1.3 mm, and circumference increased from 38.6±5.4 mm to 49.8±6.7 mm. All these changes were statistically significant (p<0.001). These findings are comparable with Li et al. (2023),[3] who established sonographic reference ranges for fetal pancreatic parameters and found progressive enlargement of the fetal pancreas with gestational age. Gilboa et al. (2021),[1] also reported that fetal pancreatic circumference correlated significantly with gestational age, abdominal circumference and estimated fetal weight.

The present study showed significantly higher fetal pancreatic dimensions among GDM pregnancies. Pancreatic transverse diameter, thickness and circumference were significantly greater in the GDM group than in the non-GDM group (p<0.001). Increased pancreatic biometry was observed in 78.9% of GDM cases compared with 29.5% of non-GDM cases. This supports the concept that maternal hyperglycemia leads to fetal hyperinsulinemia and pancreatic enlargement. Gilboa et al. (2024),[4] reported that fetal pancreatic circumference at or above the 80th centile may act as an early marker for future GDM. Similarly, Keven et al. (2025),[5] found significantly increased fetal pancreatic circumference and percentile values in GDM pregnancies during 24–28 weeks, supporting its role as an adjunct sonographic marker.

The mean birth weight was significantly higher in the GDM group than in the non-GDM group (3.18±0.42 kg vs 2.82±0.39 kg, p=0.001). This finding is consistent with the known fetal anabolic effect of insulin in pregnancies complicated by maternal hyperglycemia. Langer et al. (2005),[6] reported that untreated or inadequately controlled GDM is associated with adverse perinatal outcomes including excessive fetal growth, neonatal complications and metabolic morbidity. Heo et al. (2024),[7] also observed that altered fetal biometric patterns in diabetic pregnancies were associated with neonatal metabolic outcomes.

In relation to FGR and adverse fetal outcomes, the present study found significantly reduced fetal pancreatic dimensions. Pancreatic circumference was 42.6±5.3 mm in the FGR/adverse outcome group compared with 51.9±6.2 mm in the normal outcome group (p<0.001). Reduced pancreatic biometry was present in 72.2% of affected pregnancies compared with 14.5% of normal pregnancies. These findings agree with Keven et al. (2025),[8] who reported that fetal pancreatic circumference and percentile values were significantly lower in late-onset FGR cases than in healthy controls. Miremberg et al (2025),[9] also demonstrated that fetal pancreatic circumference is frequently reduced in growth-restricted fetuses,

suggesting impaired pancreatic development in compromised intrauterine environments.

The present study also showed that low birth weight and NICU admission were significantly higher among pregnancies with reduced pancreatic biometry. Low birth weight was seen in 66.7% of affected cases compared with 12.9% of normal outcome cases, while NICU admission was required in 38.9% and 9.7%, respectively. These findings indicate that reduced fetal pancreatic size may reflect fetal undernutrition and placental insufficiency. Chew et al. (2024),[2] highlighted that FGR is associated with neonatal morbidity, stillbirth risk and need for close surveillance. Thus, fetal pancreatic biometry may provide additional predictive information beyond routine fetal growth measurements.

CONCLUSION

The present prospective observational study demonstrated that fetal pancreatic biometry is a promising ultrasonographic marker for the prediction of both gestational diabetes mellitus (GDM) and fetal growth restriction (FGR). Fetal pancreatic dimensions increased significantly with advancing gestational age, reflecting normal pancreatic growth. Pregnancies complicated by GDM showed significantly larger pancreatic transverse diameter, thickness, and circumference compared with non-GDM pregnancies, indicating a strong association between maternal hyperglycemia and fetal pancreatic enlargement. Conversely, pregnancies complicated by FGR and adverse fetal outcomes exhibited significantly reduced pancreatic measurements, suggesting impaired fetal growth and metabolic adaptation. Reduced fetal pancreatic biometry was also associated with low birth weight and increased NICU admissions. These findings suggest that fetal pancreatic biometry may serve as a simple, non-invasive, and clinically useful adjunct to routine obstetric ultrasonography for early identification of pregnancies at risk for GDM, FGR, and adverse perinatal outcomes.

Limitations of the Study

1. The study was conducted at a single tertiary care center, which may limit the generalizability of the findings to other populations.
2. The sample size of 80 participants was relatively small and may not fully represent the broader obstetric population.
3. Fetal pancreatic measurements are operator-dependent and may be affected by fetal position, maternal habitus, and ultrasound image quality.
4. Serial pancreatic measurements were performed within a limited gestational age range, restricting evaluation of pancreatic growth throughout the entire pregnancy.
5. Potential confounding factors such as maternal diet, glycemic control, placental function, and genetic influences on fetal growth were not assessed in detail.
6. Long-term neonatal metabolic outcomes were not evaluated, preventing assessment of the relationship between fetal pancreatic size and future metabolic health.
7. Standardized reference ranges and universally accepted cut-off values for fetal pancreatic biometry are not yet available, limiting immediate clinical applicability.

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