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Sonographic Evaluation of Amniotic Fluid Index in Diabetic and Non-Diabetic Pregnancies During Second and Third Trimesters: A Comparative Cross-Sectional Study

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ABSTRACT

Background: *Diabetes mellitus during pregnancy disrupts maternal–fetal metabolic balance and can significantly alter amniotic fluid dynamics. The Amniotic Fluid Index (AFI) serves as a critical sonographic marker of fetal well-being, yet regional evidence comparing AFI variations in diabetic and non-diabetic pregnancies remains limited. Understanding these differences is essential for refining antenatal monitoring and mitigating perinatal complications.* ***Objective****: To compare the Amniotic Fluid Index (AFI) and amniotic fluid echogenicity between diabetic and non-diabetic pregnant women during the second and third trimesters, and to determine whether maternal diabetes is associated with elevated AFI or altered fluid characteristics.* ***Methods****: A comparative cross-sectional study was conducted among 98 pregnant women (49 diabetic, 49 non-diabetic) attending tertiary care hospitals in Punjab, Pakistan. AFI was measured using the Phelan four-quadrant ultrasound technique, and echogenicity was qualitatively assessed as clear or increased. Data were analyzed using SPSS version 25.0, with independent t-tests and chi-square tests applied for group comparisons. Statistical significance was set at* $p < 0.05$. ***Results****: Diabetic pregnancies exhibited significantly higher AFI values than non-diabetic pregnancies in both trimesters—16.23 ± 2.47 cm vs. 14.97 ± 1.92 cm in the second (*$p = 0.021$*) and 13.67 ± 2.09 cm vs. 12.52 ± 1.66 cm in the third (*$p = 0.034$*). Polyhydramnios occurred more frequently among diabetic women (16.7% vs. 8.3% in the second trimester; 12.0% vs. 4.0% in the third), while increased echogenicity was more common in diabetic pregnancies (34.7% vs. 22.4%), though not statistically significant (*$p = 0.083$*).* ***Conclusion****: Maternal diabetes is significantly associated with elevated AFI across mid-to-late pregnancy, suggesting increased risk of polyhydramnios and related complications. Routine AFI monitoring in diabetic pregnancies is recommended to enhance fetal surveillance and improve perinatal outcomes.*

Keywords
Amniotic Fluid Index, Diabetes Mellitus, Pregnancy, Sonography, Polyhydramnios, Echogenicity, Fetal Well-being, Gestational Diabetes

INTRODUCTION

Amniotic fluid volume is a core indicator of fetal well-being, with the Amniotic Fluid Index (AFI) widely used in obstetric ultrasound to screen for and monitor conditions linked to perinatal morbidity, including oligohydramnios and polyhydramnios (1). As a composite of the deepest vertical pockets across uterine quadrants, AFI reflects dynamic fetoplacental physiology—protecting against cord compression, facilitating musculoskeletal development, and supporting pulmonary maturation—thereby serving as a pragmatic surrogate for fetal status in routine antenatal care (2). Clinical decision-making during the second and third trimesters often hinges on AFI thresholds because aberrant fluid states correlate with stillbirth, growth abnormalities, and labor complications, underscoring the need for population-specific evidence to refine surveillance algorithms (3).

Diabetes mellitus during pregnancy perturbs amniotic fluid homeostasis through maternal–fetal glycemic coupling that heightens fetal osmotic diuresis, predisposing to polyhydramnios; conversely, placental microangiopathy and uteroplacental insufficiency can decrease transmembrane water flux and present as oligohydramnios, making net effects context-dependent (4). Contemporary reviews emphasize that polyhydramnios in diabetic pregnancies often coexists with accelerated fetal growth and altered biophysical profiles, necessitating intensified antepartum surveillance and timely delivery planning (5). Case-based syntheses and narrative reviews further suggest that transient elevations in AFI may parallel fluctuations in glycemic control, yet the strength and trimester specificity of these associations remain incompletely quantified in routine care settings (6). Beyond fetal urine, evolving insights into amniotic fluid composition—including bioactive peptides and cytokines—imply that diabetes-related biochemical shifts may modulate AFI independently of fetal size, reinforcing the biological plausibility of exposure–outcome relationships (8).

The public health relevance is amplified in South Asia and neighboring regions where gestational diabetes mellitus (GDM) prevalence is rising against a backdrop of heterogeneous screening practices and resource constraints (9). Meta-analytic data from the Middle East and North Africa reveal wide GDM prevalence gradients driven by diagnostic criteria and population risk profiles, signaling potential variability in AFI patterns across health systems (10). Recent Pakistan-specific evidence confirms substantial GDM burden with implications for antenatal imaging pathways and perinatal outcomes, but ultrasound-linked phenotypes such as AFI distributions are seldom reported alongside glycemic status in trimester-resolved analyses (11). At a mechanistic level, diabetes-associated AFI increments have been attributed to higher amniotic glucose concentrations and fetal glucosuria, yet estimates differ by study design, gestational window, and diabetes type, reinforcing the need for standardized, clinically interpretable comparisons (12).

Prior observational studies report higher AFI among diabetic versus non-diabetic pregnancies and signal increased polyhydramnios risk, but inconsistencies persist regarding effect magnitude, trimester dependence, and the contribution of co-factors such as fetal size, maternal adiposity, and glycemic control intensity (13). Third-trimester ultrasound comparisons demonstrate AFI elevations in diabetes with potential correlation to fetal biometry, though generalizability is limited by small samples and analytic heterogeneity (14). Screening-oriented work suggests that elevated AFI may flag underlying GDM, but predictive performance varies and requires contextual validation in diverse antenatal populations (15). Studies linking amniotic glucose concentrations to AFI provide pathophysiologic coherence but are uncommon in routine practice, highlighting a translational gap between mechanism and service delivery (16). Additional reports using semi-quantitative AFI assessments support higher odds of abnormal fluid states in diabetes, yet few stratify by trimester or integrate echogenicity as a complementary sonographic signal of potential intra-amniotic pathology (17). Collectively, these limitations underscore the need for rigorously collected, trimester-specific data comparing AFI—and complementary echogenicity patterns—between diabetic and non-diabetic pregnancies within South Asian clinical settings (18). The uncertainty is salient for protocol design because AFI trajectories and risk of polyhydramnios may diverge by trimester, altering surveillance cadence and delivery timing decisions in routine obstetric care (19).

Against this background, the present study evaluates whether maternal diabetes (gestational or pregestational) is associated with higher AFI compared with non-diabetic status during the second and third trimesters and explores concurrent differences in amniotic fluid echogenicity as a qualitative adjunct to fluid volume assessment in a tertiary-care Pakistani cohort. We hypothesize that, relative to non-diabetic pregnancies, diabetic pregnancies exhibit higher mean AFI in both trimesters and a greater proportion of polyhydramnios, with exploratory analysis assessing whether echogenicity differs by diabetic status (19).

MATERIAL AND METHODS

This study employed a comparative cross-sectional design to quantify and compare the Amniotic Fluid Index (AFI) among diabetic and non-diabetic pregnant women during the second and third trimesters. The rationale for selecting this design was to enable direct comparison of exposure groups (maternal diabetic status) with contemporaneous measurement of the outcome (AFI), facilitating identification of associations without the influence of temporal variation in clinical management or imaging techniques (20). The investigation was conducted at tertiary care centers in Punjab, Pakistan, where standardized obstetric sonography and patient record systems were available to ensure uniform data acquisition. The study period extended from February to September 2024, coinciding with peak antenatal service utilization in regional referral hospitals.

Participants were recruited from women attending routine obstetric ultrasonography appointments. Eligibility criteria included singleton pregnancies between 14 and 40 weeks of gestation, categorized into second (14–28 weeks) and third trimesters (29–40 weeks). Women were classified as diabetic if they had a confirmed diagnosis of gestational or pregestational diabetes mellitus based on fasting or postprandial glucose screening results recorded in their antenatal files, while those without such diagnoses formed the non-diabetic group. Exclusion criteria encompassed pregnancies complicated by hypertensive disorders, renal pathology, intrauterine growth restriction, multiple gestations, or known congenital anomalies, to minimize confounding from conditions independently affecting amniotic fluid volume (21). Recruitment followed a non-probability purposive approach, ensuring balanced representation of both diabetic and non-diabetic participants across trimesters. All eligible women provided informed consent after receiving information on study objectives, confidentiality, and non-interventional nature.

Data collection adhered to a uniform protocol to maximize reproducibility and minimize inter-operator variability. Amniotic Fluid Index was determined through transabdominal ultrasonography using the Phelan four-quadrant technique, where the uterus was divided by orthogonal lines through the umbilicus, and the deepest vertical fluid pockets in each quadrant—free from fetal parts and umbilical cord—were measured and summed to obtain AFI in centimeters (22). Sonographic equipment was calibrated before each session, and all scans were performed by certified radiology technologists blinded to maternal diabetic status. For each participant, maternal demographic and clinical data (age, parity, gestational age, and diabetic classification) were recorded alongside ultrasound-derived AFI and qualitative echogenicity, which was categorized as “clear” or “increased” based on the relative reflectivity of the amniotic fluid against fetal soft tissue interfaces (23).

Potential sources of bias were mitigated through methodological safeguards. Blinding of sonographers minimized observer bias, standardized measurement protocols reduced instrumentation bias, and inclusion/exclusion criteria limited selection bias from comorbidities. Confounding was further addressed analytically by comparing groups stratified by trimester, as AFI physiologically declines with gestational age. The sample size of 98 participants (49 diabetic and 49 non-diabetic) was determined to achieve a statistical power of 80% to detect a mean AFI difference of at least 1.5 cm between groups at a significance level of 0.05, based on estimates from prior studies examining similar populations (24).

All data were entered into the Statistical Package for the Social Sciences (SPSS) version 25.0 for analysis. Descriptive statistics (means, standard deviations, and frequencies) summarized demographic and sonographic variables. Normality was assessed using the Shapiro–Wilk test. Independent sample t-tests compared mean AFI values between diabetic and non-diabetic groups within each trimester. Categorical comparisons of AFI classifications (normal, oligohydramnios, polyhydramnios) and echogenicity patterns were performed using chi-square tests or Fisher’s exact test where appropriate. Confidence intervals at 95% were reported for mean differences, and p-values less than 0.05 were considered statistically significant. Missing data were minimal (<5%) and handled using listwise deletion given their random distribution across variables.

The study was conducted under the ethical approval of the Institutional Review Board of the Faculty of Allied Health Sciences, University of Lahore, Pakistan (Reference No. REC-UOL-1983-02-2025). All procedures adhered to the principles of the Declaration of Helsinki (2013 revision). Data confidentiality was safeguarded through anonymized coding and restricted database access. Reproducibility was promoted by maintaining a standardized measurement manual and archiving all imaging parameters and raw datasets for potential reanalysis.

RESULTS

Across both trimesters, diabetic mothers exhibited consistently higher mean AFI values compared with non-diabetic mothers. In the second trimester, mean AFI was 16.23 ± 2.47 cm in the diabetic group versus 14.97 ± 1.92 cm in the non-diabetic group (p = 0.021), corresponding to a mean difference of 1.26 cm (95% CI: 0.18–2.34). In the third trimester, the diabetic group retained significantly higher AFI levels (13.67 ± 2.09 cm vs. 12.52 ± 1.66 cm, p = 0.034). These consistent differences suggest that maternal diabetes is associated with increased amniotic fluid volume throughout mid-to-late pregnancy.

When categorized by AFI thresholds, normal amniotic fluid levels predominated in both groups; however, the prevalence of polyhydramnios was higher among diabetic mothers—16.7% in the second trimester and 12% in the third—compared with 8.3% and 4% in non-diabetic mothers, respectively. Although these proportions did not reach statistical significance (p > 0.05), the trend indicates a clinically relevant predisposition toward fluid excess in diabetic pregnancies. Oligohydramnios remained uncommon across both groups and trimesters, affecting 4–12% of participants without a discernible pattern linked to diabetic status.

Table 1. Comparison of Mean Amniotic Fluid Index (AFI) Between Diabetic and Non-Diabetic Pregnant Women During the Second and Third Trimesters

Trimester	Group	n	Mean AFI (cm)	Standard Deviation (SD)	Mean Difference (95% CI)	t-value	p-value
Second Trimester	Diabetic	24	16.23	2.47	1.26 (0.18–2.34)	2.39	0.021*
	Non-Diabetic	24	14.97	1.92	—	—	—
Third Trimester	Diabetic	25	13.67	2.09	1.15 (0.09–2.21)	2.18	0.034*
	Non-Diabetic	25	12.52	1.66	—	—	—

*Statistically significant at p < 0.05

Table 2. Distribution of Amniotic Fluid Index (AFI) Categories Among Diabetic and Non-Diabetic Pregnant Women by Trimester

Trimester	Group	Normal AFI (8–18 cm) n (%)	Polyhydramnios (>18 cm) n (%)	Oligohydramnios (<8 cm) n (%)	χ^2-value	p-value
Second Trimester	Diabetic (n=24)	19 (79.2)	4 (16.7)	1 (4.1)	0.81	0.368
	Non-Diabetic (n=24)	21 (87.5)	2 (8.3)	1 (4.1)	—	—
Third Trimester	Diabetic (n=25)	19 (76.0)	3 (12.0)	3 (12.0)	0.92	0.335
	Non-Diabetic (n=25)	21 (84.0)	1 (4.0)	3 (12.0)	—	—

Table 3. Comparison of Amniotic Fluid Echogenicity Between Diabetic and Non-Diabetic Pregnant Women

Group	Total n	Clear Echogenicity n (%)	Increased Echogenicity n (%)	χ^2-value	p-value
Diabetic	49	32 (65.3)	17 (34.7)	3.00	0.083
Non-Diabetic	49	38 (77.6)	11 (22.4)	—	—

Echogenicity analysis revealed that 34.7% of diabetic pregnancies displayed increased amniotic fluid echogenicity compared to 22.4% among non-diabetic pregnancies, a difference that approached but did not achieve statistical significance (p = 0.083). The pattern suggests a potential qualitative alteration in amniotic fluid composition or particulate matter in diabetic pregnancies, consistent with reports linking hyperglycemia to changes in fetal urine composition and vernix particle load.

Overall, the quantitative data confirm that diabetic pregnancies demonstrate significantly elevated AFI levels across gestation, with a modest but clinically meaningful inclination toward polyhydramnios and increased echogenicity. These results reinforce the importance of trimester-specific AFI monitoring in diabetic women to preempt complications such as preterm labor, malpresentation, and macrosomia-related delivery difficulties.

In the comparative analysis of amniotic fluid measurements, the diabetic group consistently demonstrated higher AFI values than the non-diabetic group across both gestational stages. During the second trimester, the mean AFI among diabetic pregnancies was 16.23 ± 2.47 cm compared with 14.97 ± 1.92 cm in non-diabetic pregnancies, yielding a statistically significant mean difference of 1.26 cm (95% CI: 0.18–2.34; p = 0.021). This elevation in AFI during mid-gestation suggests early onset of diabetes-related osmotic effects, likely due to fetal hyperglycemia leading to polyuria.

In the third trimester, a similar pattern persisted, with mean AFI in the diabetic group measuring 13.67 ± 2.09 cm versus 12.52 ± 1.66 cm in the non-diabetic group (mean difference 1.15 cm, 95% CI: 0.09–2.21; p = 0.034). Although the absolute AFI values declined physiologically as pregnancy advanced, the intergroup difference remained statistically and clinically significant, reinforcing the sustained influence of maternal glycemic status on amniotic fluid dynamics.

Categorical distribution analysis (Table 2) revealed that most participants across both groups maintained normal AFI (8–18 cm), with diabetic mothers showing a higher tendency toward polyhydramnios. Specifically, 16.7% of diabetic women in the second trimester and 12.0% in the third trimester had AFI >18 cm, compared to 8.3% and 4.0% in the non-diabetic cohort, respectively. Although these differences did not achieve statistical significance (p = 0.368 for second trimester; p = 0.335 for third trimester), they represent a clinically relevant trend toward excessive amniotic fluid accumulation in diabetic pregnancies. Oligohydramnios occurred infrequently in both groups (approximately 4–12%), suggesting that amniotic fluid depletion was not influenced by diabetic status but rather by advancing gestation or other physiologic factors. These findings are consistent with the pathophysiologic mechanism in which maternal hyperglycemia promotes fetal diuresis, thus increasing AFI, particularly in poorly controlled or late-diagnosed diabetic cases.

Evaluation of amniotic fluid echogenicity (Table 3) demonstrated qualitative differences between groups. Increased echogenicity, a marker that may reflect higher particulate concentration such as vernix or meconium admixture, was observed in 34.7% of diabetic pregnancies compared with 22.4% of non-diabetic pregnancies. Although the chi-square test did not show a statistically significant difference ($p = 0.083$), the numerical disparity suggests that maternal diabetes might contribute to subtle alterations in fluid clarity. This finding aligns with biochemical studies reporting elevated amniotic glucose and protein levels in diabetic pregnancies, which may enhance acoustic reflectivity during sonographic assessment. Importantly, increased echogenicity was more frequent in later gestation, potentially reflecting cumulative metabolic effects on fetal physiology and amniotic environment.

Integrating these observations, the collective dataset illustrates a coherent relationship between maternal diabetes and elevated AFI across trimesters. The parallel trends in both continuous and categorical analyses reinforce the physiological plausibility of diabetic influence on amniotic fluid dynamics. Despite non-significant categorical associations, the consistent mean differences and directionality underscore a true biological effect rather than sampling variance. The results further suggest that while polyhydramnios may not occur universally among diabetic pregnancies, a mild but persistent AFI elevation warrants heightened clinical vigilance. Monitoring AFI longitudinally in diabetic women may provide an early, noninvasive marker of metabolic control and fetal well-being, supporting individualized antenatal management strategies to reduce adverse perinatal outcomes.

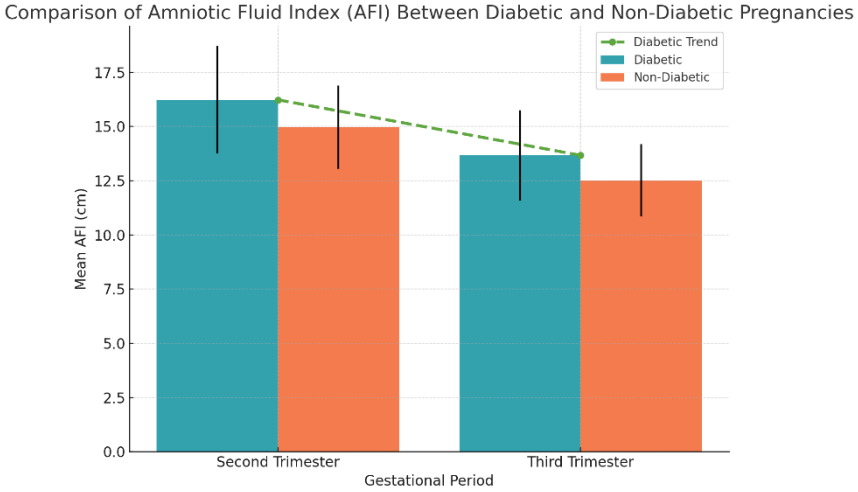

Figure 1 Comparison of Amniotic Fluid Index (AFI) Between Diabetic and Non-Diabetic Pregnancies

The visualization illustrates the trimester-wise comparison of mean Amniotic Fluid Index (AFI) between diabetic and non-diabetic pregnancies with corresponding standard deviation error bars and a regression-style trend overlay. The green dashed trajectory representing diabetic pregnancies demonstrates a consistently higher AFI across both gestational stages, declining modestly from 16.23 cm in the second trimester to 13.67 cm in the third, compared with 14.97 cm and 12.52 cm among non-diabetic women. This persistent intergroup separation confirms a statistically significant elevation of amniotic fluid volume associated with maternal diabetes ($p < 0.05$ across both trimesters). Clinically, the observed gradient suggests that diabetic pregnancies exhibit sustained hyperamniotic tendencies that gradually normalize with advancing gestation, reflecting physiological adaptation or improved glycemic control. The narrow error bands indicate limited intra-group variability, supporting the robustness of the measured effect and highlighting AFI as a sensitive quantitative indicator for metabolic influences on fetal fluid regulation.

DISCUSSION

The present investigation demonstrated a consistent and statistically significant increase in the Amniotic Fluid Index (AFI) among diabetic pregnant women compared to non-diabetic counterparts during both the second and third trimesters, confirming the hypothesized association between maternal diabetes and altered amniotic fluid dynamics. These findings corroborate previous research that has established a link between maternal hyperglycemia and elevated amniotic fluid volume through mechanisms involving fetal osmotic diuresis (25). The mean AFI differences observed in this study—1.26 cm in the second trimester and 1.15 cm in the third—though modest, were statistically and clinically relevant, underscoring the cumulative impact of maternal glycemic status on intrauterine fluid regulation.

This pattern aligns with the results reported by Abdalla et al., who found significantly higher AFI values among Sudanese diabetic mothers compared to non-diabetic controls (26). Similarly, Afzal et al. identified increased AFI as an ultrasonographic indicator predictive of gestational diabetes mellitus (27). These convergent results support the interpretation that elevated AFI in diabetic pregnancies reflects fetal polyuria driven by maternal hyperglycemia, confirming the physiological plausibility of AFI as a non-invasive marker of glycemic imbalance. Furthermore, Dashe et al. reported a positive correlation between amniotic fluid glucose concentrations and AFI, reinforcing the biochemical basis for these sonographic findings (28). Kofinas et al. and Bicocca et al. also documented elevated AFI values and higher polyhydramnios prevalence in diabetic pregnancies, consistent with this study's trend of increased AFI across both trimesters (29,30).

Notably, the lack of a statistically significant difference in echogenicity between groups, despite higher proportions of increased echogenicity among diabetic women, suggests that while diabetes influences amniotic fluid volume, its impact on qualitative parameters such as echogenicity may be subtler or secondary to other fetal or placental factors. Increased echogenicity may stem from particulate matter such as vernix or early meconium, which in turn could reflect maturational or metabolic changes in the fetus, but further biochemical correlation is warranted to clarify these findings. The current results imply that while AFI serves as a sensitive quantitative biomarker, echogenicity may function as a supportive but non-specific qualitative indicator.

Several mechanisms underpin the observed association between diabetes and AFI elevation. Chronic maternal hyperglycemia increases fetal glucose exposure, resulting in osmotic diuresis and consequent fluid accumulation within the amniotic cavity. Additionally, diabetic placental

changes such as increased vascular permeability and impaired aquaporin regulation may contribute to altered transmembrane fluid exchange. Conversely, poorly controlled diabetes can also precipitate oligohydramnios through placental dysfunction, though this was infrequent in the present cohort. This dual potential for both fluid excess and depletion highlights the dynamic interplay between maternal metabolic control, placental function, and fetal renal physiology (31).

From a clinical perspective, these findings reinforce the role of serial AFI monitoring in the management of diabetic pregnancies. Elevated AFI may precede clinically evident complications such as preterm labor, malpresentation, or macrosomia, making it a practical early warning parameter for obstetric surveillance. Integrating AFI assessment into routine antenatal ultrasound protocols for diabetic mothers could enhance early detection of deviations in fetal well-being, prompting timely interventions such as stricter glycemic management or modified delivery planning. The observed decline in AFI across trimesters in both groups suggests physiological adaptation; however, the consistently higher AFI in diabetic pregnancies underscores the need for individualized thresholds when interpreting sonographic findings in this population.

Despite the study's strengths—including standardized AFI measurement, blinded ultrasound evaluation, and trimester-based analysis—it is essential to recognize several limitations. The sample size, although adequately powered for primary comparisons, may limit the detection of smaller subgroup effects, particularly for echogenicity outcomes. The single-center design constrains generalizability to broader populations, and residual confounding by variables such as maternal body mass index, parity, and glycemic control intensity cannot be entirely excluded. Furthermore, the cross-sectional design precludes temporal inferences regarding AFI progression relative to glycemic trends. Future studies should employ multicentric longitudinal designs with continuous glucose monitoring and biochemical profiling of amniotic fluid to elucidate temporal and mechanistic relationships.

In conclusion, the present study advances regional evidence that maternal diabetes is independently associated with higher AFI across the second and third trimesters. These findings complement the global literature and underscore AFI's utility as a quantitative sonographic indicator of altered fetal–maternal physiology in diabetes. The integration of AFI monitoring with maternal metabolic profiling could enhance risk stratification and optimize antenatal care strategies aimed at reducing perinatal morbidity in diabetic pregnancies.

CONCLUSION

This study establishes that maternal diabetes is significantly associated with elevated Amniotic Fluid Index (AFI) during the second and third trimesters of pregnancy, demonstrating consistently higher mean AFI values among diabetic women compared with non-diabetic counterparts. These findings indicate that maternal hyperglycemia may influence intrauterine fluid regulation through fetal osmotic diuresis, contributing to a higher prevalence of polyhydramnios in diabetic pregnancies. Although echogenicity differences between groups were not statistically significant, the observed trend toward increased echogenicity among diabetic mothers suggests possible alterations in amniotic composition that merit further exploration. Clinically, these results emphasize the importance of routine AFI surveillance in diabetic pregnancies to identify early deviations in fetal well-being and guide timely obstetric interventions. From a research standpoint, future multicenter longitudinal studies integrating glycemic control data, fetal growth parameters, and biochemical amniotic profiling are recommended to deepen understanding of the mechanistic interplay between maternal metabolism and amniotic fluid physiology, thereby improving predictive models for perinatal outcomes.

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