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Ultrasound Evaluation of Fetal Growth Patterns in Antenatal Women with Gestational Diabetes Mellitus

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Abstract:

Background: Gestational Diabetes Mellitus (GDM) is a major metabolic complication of pregnancy and is strongly associated with fetal overgrowth and large-for-gestational-age (LGA) infants. Even mild maternal hyperglycemia alters fetal fat deposition, increasing the risk of macrosomia and related perinatal complications including shoulder dystocia, neonatal hypoglycemia, operative delivery, and birth trauma. Serial ultrasonographic assessment of fetal biometry plays a critical role in identifying accelerated growth patterns and guiding clinical management.

Methods: This hospital-based case–control study included 150 antenatal women between 28–40 weeks of gestation, comprising 75 women diagnosed with GDM using DIPSI criteria and 75 normoglycemic controls. Maternal demographic and metabolic characteristics were recorded. Fetal biometric parameters—biparietal diameter, head circumference, abdominal circumference, femur length, and estimated fetal weight—were measured and plotted against WHO growth standards. Fetuses were categorized as small-, appropriate-, or large-for-gestational-age. Among GDM mothers, treatment modality (meal plan, oral hypoglycemic agents [OHA], or insulin) and glycemic control (FBS and PPBS levels) were analyzed. Logistic regression models were applied to identify independent predictors of LGA.

Results: Women with GDM had significantly higher pre-pregnancy BMI and a greater prevalence of family history of diabetes and previous GDM. Fetuses of GDM mothers demonstrated significantly higher abdominal circumference and estimated fetal weight compared to controls. LGA prevalence was nearly threefold higher in GDM pregnancies (29.3%) compared to normoglycemic pregnancies (8.0%). Among GDM mothers, LGA occurred in 14.3% of diet-controlled cases compared to 37.5% in the OHA group and 39.1% in the insulin group (p=0.002). Mothers requiring pharmacologic therapy had higher mean FBS (98–104 mg/dL) and PPBS (142–158 mg/dL), demonstrating nearly a threefold increase in LGA risk with suboptimal glycemic control. Serial growth assessment revealed that significant divergence in abdominal circumference and fetal weight became evident after 32 weeks of gestation. On multivariate analysis, current GDM, elevated pre-pregnancy BMI, and increased fetal abdominal circumference remained independent predictors of LGA.

Conclusion: Gestational diabetes significantly increases the risk of fetal overgrowth, particularly when glycemic control is suboptimal. Elevated pre-pregnancy BMI independently contributes to LGA risk, underscoring the importance of preconception weight optimization and early metabolic screening. Strict glycemic control and serial third-trimester ultrasound monitoring—especially fetal abdominal circumference tracking—are essential strategies to reduce LGA-associated perinatal complications.

Keywords: Gestational Diabetes Mellitus; Large for Gestational Age; Fetal Abdominal Circumference; Glycemic Control; Pre-pregnancy Body Mass Index; Prenatal Ultrasonography

Introduction: Gestational Diabetes Mellitus (GDM) is defined as glucose intolerance of varying severity first recognized during pregnancy and is one of the most common metabolic complications of gestation. Pregnancy induces progressive insulin resistance mediated by placental hormones, including human placental lactogen, progesterone, cortisol, and growth hormone. When pancreatic β-cell compensation becomes inadequate, maternal hyperglycemia develops, resulting in GDM [1]. Maternal hyperglycemia facilitates transplacental glucose transfer, stimulating fetal pancreatic β-cell hyperplasia and hyperinsulinemia, which promotes accelerated somatic growth and adiposity, particularly in the abdominal region [2].Globally, GDM is increasingly recognized as a significant public health challenge. Changing diagnostic thresholds, rising maternal age, increasing obesity prevalence, and sedentary lifestyles have contributed to its expanding burden [3]. In India, GDM has emerged as a major concern due to genetic predisposition, high background prevalence of type 2 diabetes, and rapid nutritional transition [4]. A recent systematic review and meta-analysis from India reported substantial regional variation in GDM prevalence, with rising trends over the past decade, reflecting both improved screening practices and genuine epidemiological transition [5]. Additionally, the Indian phenotype—characterized by higher visceral adiposity at lower body mass index (BMI)—contributes to increased insulin resistance during pregnancy [6].

According to the International Diabetes Federation, hyperglycemia in pregnancy affects approximately one in seven live births globally, with a disproportionately higher burden in low- and middle-income countries [7]. Advancing maternal age has consistently been shown to increase GDM risk, with meta-analytic evidence demonstrating a stepwise rise in incidence across age categories [8]. Hospital-based Indian studies further confirm strong associations between elevated BMI, family history of diabetes, and development of GDM [9]. Lifestyle factors, including dietary patterns and reduced physical activity, also play a significant role in modulating risk [10].

Recent institutional data from tertiary centers in India indicate increasing detection rates of GDM, partly attributable to universal screening approaches [11]. Population-based cohort studies have demonstrated that GDM is associated not only with maternal metabolic dysfunction but also with adverse perinatal outcomes, including fetal overgrowth and neonatal complications [12]. However, differences in diagnostic criteria—particularly between IADPSG and DIPSI approaches—continue to influence reported prevalence estimates and complicate inter-study comparisons [13].Analysis of National Family Health Survey (NFHS) data reveals significant regional disparities in GDM prevalence across India, influenced by socioeconomic status, urbanization, BMI distribution, and healthcare access [14]. Tamil Nadu, a state with high background diabetes prevalence and effective antenatal screening programs, reports relatively elevated GDM rates, underscoring the importance of region-specific evaluation of maternal and fetal outcomes.

One of the most clinically significant consequences of GDM is fetal overgrowth. Large-for-gestational-age (LGA) infants are at increased risk of shoulder dystocia, birth trauma, neonatal hypoglycemia, operative delivery, and long-term metabolic sequelae. Excess intrauterine glucose exposure leads to disproportionate fat deposition, particularly reflected by increased fetal abdominal circumference and estimated fetal weight. Ultrasonographic assessment of fetal biometry therefore plays a central role in monitoring pregnancies complicated by GDM. Among biometric parameters, abdominal circumference is considered a sensitive marker of fetal adiposity and metabolic influence.

Despite growing literature on GDM prevalence in India, limited data exist from South India examining the combined influence of pre-pregnancy BMI, family history of diabetes, previous GDM, glycemic control, and serial fetal growth progression on LGA outcomes. Furthermore, the relationship between treatment modality (diet control, oral hypoglycemic agents, or insulin therapy) and fetal growth patterns remains underexplored in regional populations.

Given the rising metabolic burden and the potential for preventable perinatal complications, it is essential to identify independent predictors of LGA in GDM pregnancies. Understanding how maternal metabolic risk factors interact with dynamic fetal growth parameters may facilitate earlier risk stratification, guide preconception counselling, optimize glycemic control strategies, and improve obstetric decision-making.

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Methods: This prospective hospital-based case–control study was conducted over a period of three months at a tertiary care center. A total of 150 antenatal women between 28 and 40 weeks of gestation were enrolled. Seventy-five women diagnosed with gestational diabetes mellitus (GDM) based on the DIPSI single-step 75-g oral glucose tolerance test formed the case group, while 75 normoglycemic pregnant women with normal DIPSI results served as controls. Controls were matched to cases for maternal age category and gestational age (±1 week) to minimize physiological variation in fetal growth. Pregnant women aged 18–40 years with singleton gestation booked and coming for regular antenatal checkups were included after obtaining written informed consent. Women with pre-existing type 1 or type 2 diabetes mellitus, multiple gestation, fetal congenital anomalies, thyroid disorders, renal or hepatic disease, and hypertensive disorders of pregnancy were excluded to reduce confounding influences on fetal growth. Sample size was calculated using the formula for comparison of two proportions, assuming 20% macrosomia prevalence in GDM and 5% in controls, with 95% confidence level and 80% power. The minimum required sample size was 75 participants per group. Maternal demographic and clinical data were recorded, including age, pre-pregnancy body mass index (BMI), gravidity, family history of diabetes, previous history of GDM, and blood pressure. Glycemic parameters such as fasting blood sugar (FBS) and postprandial blood sugar (PPBS) were monitored in the GDM group. Among women with GDM, treatment modality was categorized as diet-controlled (meal plan), oral hypoglycemic agents (OHA), or insulin therapy.

Ultrasonographic examination was performed using a standardized protocol with a high-resolution transabdominal probe. Fetal biometric measurements included biparietal diameter (BPD), head circumference (HC), abdominal circumference (AC), and femur length (FL). Estimated fetal weight (EFW) was calculated using Hadlock's formula. Serial fetal growth assessments were performed at 28, 32, and 36 weeks of gestation. Growth categories were determined using WHO reference standards and classified as small-for-gestational-age (SGA), appropriate-for-gestational-age (AGA), or large-for-gestational-age (LGA).

The primary outcome was the occurrence of LGA fetus. Continuous variables were expressed as mean ± standard deviation and compared using independent t-tests. Categorical variables were compared using chi-square tests. Univariate logistic regression was performed to identify factors associated with LGA, followed by multivariate logistic regression to determine independent predictors. Odds ratios (OR) and adjusted odds ratios (AOR) were reported with 95% confidence intervals. A p-value <0.05 was considered statistically significant.

The study was approved by the Institutional Ethics Committee. Written informed consent was obtained from all participants, and confidentiality was maintained throughout the study.

Results: The study included 150 antenatal women, comprising 75 with gestational diabetes mellitus and 75 normoglycemic controls. Baseline characteristics were compared between the groups, followed by evaluation of fetal biometric parameters and growth categories. Subsequent analyses assessed the distribution of small, appropriate, and large-for-gestational-age fetuses. Maternal and fetal predictors of large-for-gestational-age outcomes were examined using univariate and multivariate models. The key findings from these comparisons are summarized below.

Figure 1: Baseline Maternal Characteristics of Study Participants (N=150)

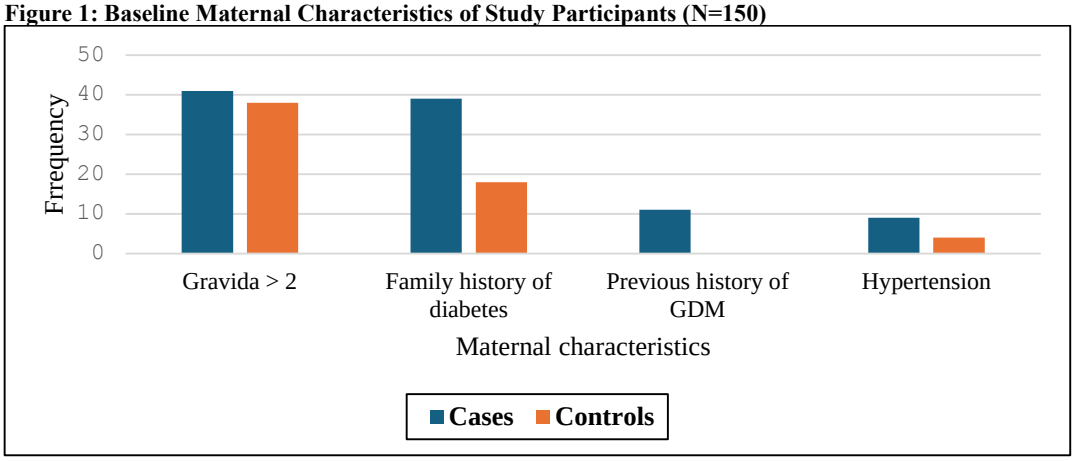

Table 1: Baseline Maternal Characteristics of Study Participants (N=150)

Variable	GDM (n=75) Mean ± SD	Controls (n=75) Mean ± SD	p-value
Maternal Age (years)	28.6 ± 4.1	27.9 ± 4.3	0.321
Pre-pregnancy BMI (kg/m²)	26.8 ± 3.9	24.1 ± 3.2	0.001
Gestational Age at Scan (weeks)	32.1 ± 2.8	32.4 ± 2.6	0.483
	Frequency n (%)	**Frequencyn (%)**	
Gravida ≥2	41 (54.7%)	38 (50.7%)	0.626
Family History of Diabetes	39 (52.0%)	18 (24.0%)	0.001
Previous GDM	11 (14.7%)	0 (0%)	<0.001
Hypertension	9 (12.0%)	4 (5.3%)	0.149

In this study involving 150 antenatal women, the baseline characteristics of those with gestational diabetes mellitus (GDM) were compared with normoglycemic controls. The mean maternal age was comparable between the two groups (28.6 vs. 27.9 years, p = 0.321), indicating no significant age difference. However, women with GDM had a **significantly higher pre-pregnancy BMI** compared to controls (26.8 vs. 24.1 kg/m², p = 0.001), suggesting that increased maternal adiposity is a potential risk factor for developing GDM.

Gestational age at the time of ultrasound assessment did not differ significantly between the groups (p = 0.483), indicating that both cohorts were evaluated at similar stages of pregnancy. Parity distribution was also comparable, with no significant difference in the proportion of women who were gravida ≥2 (p = 0.626).A **notable difference** was observed in the prevalence of **family history of diabetes**, which was significantly more common in the GDM group (52.0% vs. 24.0%, p = 0.001), highlighting the role of genetic predisposition. Additionally, **previous history of GDM** was reported only among the GDM group (14.7% vs. 0%), demonstrating a strong recurrence tendency (p < 0.001). Although a higher proportion of women with GDM had hypertension (12.0% vs. 5.3%), this difference was not statistically significant (p = 0.149). Overall, the results indicate that **higher BMI, family history of diabetes, and prior GDM** are important baseline characteristics significantly associated with the development of gestational diabetes. (Figure 1 & Table 1)

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Table 2: Comparison of Fetal Biometric Parameters Between Groups (N=150)

Parameter	GDM (Mean ± SD)	Controls (Mean ± SD)	p-value
Biparietal Diameter (cm)	8.21 ± 0.44	8.04 ± 0.40	0.033
Head Circumference (cm)	30.1 ± 1.9	29.6 ± 1.8	0.061
Abdominal Circumference (cm)	29.4 ± 2.8	27.1 ± 2.5	<0.001
Femur Length (cm)	6.28 ± 0.46	6.13 ± 0.43	0.072
Estimated Fetal Weight (g)	2240 ± 410	2015 ± 380	<0.001

Comparison of fetal biometric parameters between GDM and non-GDM pregnancies revealed notable differences in growth patterns. The mean biparietal diameter (BPD) was significantly higher among fetuses of mothers with GDM compared to controls (8.21 ± 0.44 cm vs. 8.04 ± 0.40 cm, p = 0.033), indicating a modest but statistically significant increase in head width. Although head circumference (HC) was also higher in the GDM group (30.1 ± 1.9 cm vs. 29.6 ± 1.8 cm), the difference did not reach statistical significance (p = 0.061).A prominent finding was the **significantly larger abdominal circumference (AC)** in fetuses of women with GDM (29.4 ± 2.8 cm vs. 27.1 ± 2.5 cm, p < 0.001). Since AC is closely associated with fetal adiposity and the metabolic effects of hyperinsulinemia, this suggests accelerated fat deposition and growth in GDM pregnancies.Femur length (FL) values were slightly higher in the GDM group (6.28 ± 0.46 cm vs. 6.13 ± 0.43 cm), but this difference was not statistically significant (p = 0.072). In contrast, **estimated fetal weight (EFW)** showed a marked and statistically significant increase in the GDM group (2240 ± 410 g vs. 2015 ± 380 g, p < 0.001), further supporting the trend of fetal overgrowth. Overall, these results indicate that fetuses in GDM pregnancies tend to have **higher abdominal circumference and estimated fetal weight**, consistent with the known risk of macrosomia in diabetic pregnancies. The pattern reflects the metabolic influence of maternal hyperglycemia on fetal growth. (Table 2)

Figure 2: Distribution of Fetal Growth Categories (N=150)

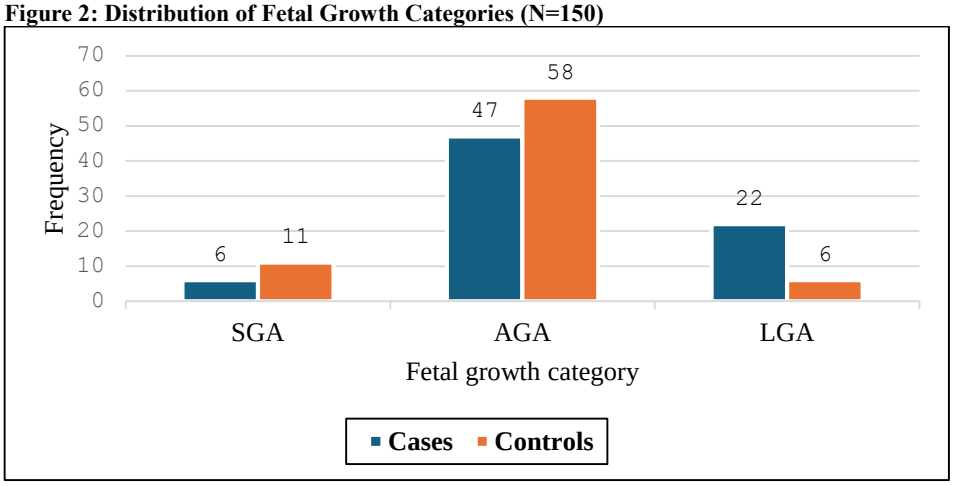

Table 3: Distribution of Fetal Growth Categories (N=150)

Growth Category	GDM (n=75) n (%)	Controls (n=75) n (%)	p-value
SGA	6 (8.0%)	11 (14.7%)	0.191
AGA	47 (62.7%)	58 (77.3%)	0.041
LGA	22 (29.3%)	6 (8.0%)	0.001

Analysis of fetal growth categories revealed distinct differences between gestational diabetes mellitus (GDM) pregnancies and normoglycemic controls. The proportion of small-for-gestational-age (SGA) fetuses was lower in the GDM group compared to controls (8.0% vs. 14.7%), although this difference was not statistically significant (p = 0.191). This suggests that growth restriction is not a predominant pattern among fetuses of mothers with GDM. A significantly smaller proportion of appropriate-for-gestational-age (AGA) fetuses was observed in the GDM group (62.7%) compared to the control group (77.3%) (p = 0.041). This reflects a shift away from normal growth patterns in GDM pregnancies. (Figure 2)Most notably, the prevalence of large-for-gestational-age (LGA) fetuses was **markedly higher** among women with GDM (29.3%) compared to controls (8.0%), and this difference was highly significant (p = 0.001). This finding aligns with the established association between maternal hyperglycemia and accelerated fetal growth, likely driven by excess fetal insulin production. Overall, the distribution of growth categories indicates a clear tendency toward **fetal overgrowth (LGA)** in GDM pregnancies, reinforcing the importance of glycemic control and close fetal monitoring in this population. (Table 3)

Table 4: Univariate Logistic Regression for Predictors of LGA (N=150)

Variable	OR (95% CI)	p-value
Maternal BMI		
Normal (<25)	Ref	
Overweight (25–29.9)	2.34 (1.01–5.41)	0.047
Obese (≥30)	4.86 (1.92–12.30)	<0.001
Family History of Diabetes		
Yes	1.98 (1.02-3.82)	
No	Ref	0.04
Previous GDM		
Yes	2.80 (1.09-7.21)	
No	Ref	0.032
Gestational Diabetes		
Yes	4.94 (2.09-11.66)	
No	Ref	<0.001
Abdominal Circumference		
Higher	1.35 (1.20-1.52)	
Lower	Ref	<0.001
Estimated Fetal Weight		
Higher EFW	1.22 (1.10-1.35)	
Low EFW	Ref	<0.001

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Univariate logistic regression analysis identified several maternal and fetal factors significantly associated with the likelihood of delivering a large-for-gestational-age (LGA) infant. Compared to women with normal pre-pregnancy BMI, overweight women had 2.34 times higher odds of delivering an LGA infant (95% CI 1.01–5.41; p = 0.047), while obese women had a markedly higher risk, with 4.86-fold increased odds (95% CI 1.92–12.30; p < 0.001). This demonstrates a graded increase in LGA risk with rising maternal adiposity.A family history of diabetes was also significantly associated with LGA, nearly doubling the odds (OR 1.98; 95% CI 1.02–3.82; p = 0.04). Similarly, women with a previous history of GDM were almost three times more likely to deliver an LGA infant (OR 2.80; 95% CI 1.09–7.21; p = 0.032), indicating persistence of metabolic vulnerability across pregnancies. Current gestational diabetes demonstrated the strongest association, increasing the odds of LGA nearly fivefold (OR 4.94; 95% CI 2.09–11.66; p < 0.001), underscoring the central role of maternal hyperglycemia in fetal overgrowth. Among fetal biometric parameters, both abdominal circumference and estimated fetal weight were significantly associated with LGA. Higher abdominal circumference increased the odds of LGA by 35% per unit increment (OR 1.35; 95% CI 1.20–1.52; p < 0.001), while higher estimated fetal weight increased the risk by 22% per incremental unit (OR 1.22; 95% CI 1.10–1.35; p < 0.001). These findings reflect the direct metabolic influence of maternal glycemic status on fetal adiposity and growth acceleration.Overall, the univariate analysis indicates that maternal metabolic factors (BMI category, family history of diabetes, prior GDM, and current GDM) as well as fetal biometric indicators (abdominal circumference and estimated fetal weight) are significantly associated with increased LGA risk. (Table 4)

Table 5: Multivariate Logistic Regression for Independent Predictors of LGA (N=150)

Predictor	Adjusted Odds Ratio (AOR)	95% CI	p-value
Gestational Diabetes			
Yes	3.92	1.55–9.88	0.004
No	Ref		
Maternal BMI (per unit increase)	1.10	1.01–1.20	0.034
Family History of Diabetes			
Yes	1.44	0.65–3.22	0.379
No	Ref		
Previous GDM			
Yes	1.96	0.65–5.90	0.232
No	Ref		
Abdominal Circumference (per cm increase)	1.22	1.07–1.40	0.003
Estimated Fetal Weight (per 100 g increase)	1.11	0.98–1.26	0.10

Multivariate logistic regression analysis was conducted to determine independent predictors of large-for-gestational-age (LGA) infants after adjustment for relevant maternal and fetal variables. Gestational diabetes mellitus remained the strongest independent determinant of LGA, with affected mothers demonstrating nearly a fourfold increased risk compared to normoglycemic women (AOR 3.92; 95% CI 1.55–9.88; p = 0.004). This confirms that active maternal hyperglycemia exerts a dominant influence on fetal overgrowth even after accounting for other metabolic and sonographic factors.

Maternal pre-pregnancy body mass index independently contributed to LGA risk. Each unit increase in BMI was associated with a 10% rise in the adjusted odds of LGA (AOR 1.10; 95% CI 1.01–1.20; p = 0.034), underscoring the independent role of maternal adiposity beyond glucose intolerance alone.

Among fetal biometric parameters, abdominal circumference emerged as a significant independent predictor. Each centimeter increase in abdominal circumference increased the odds of LGA by 22% (AOR 1.22; 95% CI 1.07–1.40; p = 0.003), highlighting its sensitivity as a marker of metabolically mediated fetal adiposity.

In contrast, family history of diabetes (p = 0.379), previous GDM (p = 0.232), and estimated fetal weight (p = 0.100) did not retain statistical significance in the adjusted model. This suggests that their apparent associations in univariate analysis are likely mediated through current glycemic status, maternal BMI, or abdominal growth parameters rather than representing independent predictors.

Taken together, the multivariate findings demonstrate that **gestational diabetes, elevated maternal BMI, and increased fetal abdominal circumference** are the key independent determinants of LGA. These results reinforce the need for focused glycemic management and close fetal growth monitoring in pregnancies complicated by GDM. (Table 5)

Table 6: Glycemic Control and Treatment Modality Among GDM Mothers and LGA Outcome (n = 75)

Treatment Modality	Mean FBS (mg/dL)	Mean PPBS (mg/dL)	LGA n (%)	AGA/SGA n (%)	p-value
Meal plan only (n=28)	88 ± 9	118 ± 14	4 (14.3)	24 (85.7)	
OHA (n=24)	98 ± 11	142 ± 18	9 (37.5)	15 (62.5)	
Insulin (n=23)	104 ± 13	158 ± 22	9 (39.1)	14 (60.9)	**0.002**

A significant association was observed between treatment modality, glycemic levels, and LGA outcome (p = 0.002). Mothers managed with meal plan alone had lower mean FBS and PPBS values and the lowest LGA rate (14.3%). In contrast, women requiring OHA and insulin had progressively higher glucose levels and markedly increased LGA proportions (37.5% and 39.1%). This trend suggests that severity of hyperglycemia, rather than therapy type alone, drives fetal overgrowth. (Table 6)

Table 7: Serial Fetal Growth Parameters at 28, 32, and 36 Weeks in GDM and Control Groups (N=150)

Group	AC (cm) Mean ± SD	EFW (g) Mean ± SD	p-value
28 weeks			
GDM	24.1 ± 2.1	1150 ± 180	0.08
Control	23.5 ± 2.0	1080 ± 170	
32 weeks			
GDM	29.4 ± 2.8	2240 ± 410	<0.001
Control	27.1 ± 2.5	2015 ± 380	
36 weeks			
GDM	33.8 ± 3.1	3480 ± 420	<0.001
Control	31.2 ± 2.9	2960 ± 390	

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At 28 weeks, abdominal circumference and estimated fetal weight were comparable between groups. However, significant divergence emerged at 32 weeks and widened further by 36 weeks ($p < 0.001$), with GDM fetuses demonstrating higher AC and EFW values. This progressive separation indicates third-trimester acceleration of metabolically mediated adipose deposition. The findings highlight the importance of serial late-pregnancy ultrasound surveillance in GDM pregnancies. (Table 7)

Figure 3: Estimated Fetal Weight Distribution Among LGA Infants (n = 28)

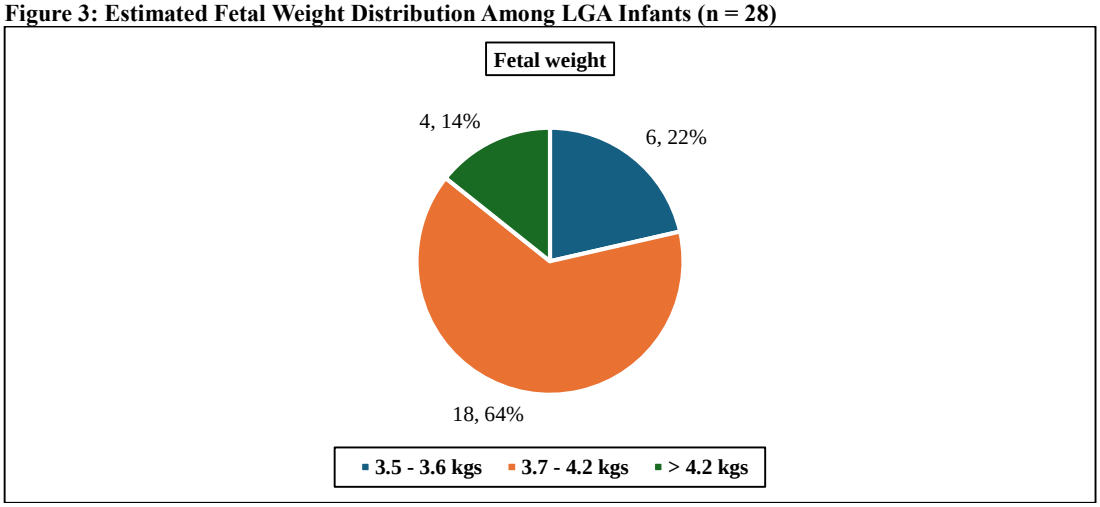

Table 8: Estimated Fetal Weight Distribution Among LGA Infants (n = 28)

EFW Range (kg)	Frequency	Percentage
3.5–3.6	6	21.4
3.7–4.2	**18**	**64.3**
>4.2	4	14.3

Among the 28 LGA infants identified, most (64.3%) had estimated fetal weights between 3.7–4.2 kg. A smaller proportion (21.4%) ranged between 3.5–3.6 kg, while 14.3% exceeded 4.2 kg, consistent with overt macrosomia. The distribution reflects substantial fetal overgrowth, with a notable fraction reaching higher weight thresholds associated with increased perinatal risk. (Figure 3 & Table 8)

Discussion: The present study demonstrates a substantial burden of fetal overgrowth among pregnancies complicated by gestational diabetes mellitus. LGA occurred in **29.3% of GDM pregnancies compared with 8.0% in normoglycemic controls ($p = 0.001$)**, translating to nearly a **3.6-fold higher prevalence**. Even after adjustment for confounders, GDM independently increased LGA risk almost fourfold (**aOR 3.92; 95% CI 1.55–9.88**). These findings reinforce that maternal hyperglycemia remains the principal determinant of excessive fetal growth in contemporary obstetric practice. The increasing metabolic burden of pregnancy in India provides an important contextual background. Rising GDM prevalence across Indian populations has been documented recently, reflecting nutritional transition and increasing maternal adiposity [15]. While that study focused on epidemiological trends, our findings demonstrate the tangible fetal consequence of this metabolic shift — namely, a markedly elevated LGA rate in affected pregnancies.

Fetal overgrowth in GDM is increasingly understood as a reflection of the broader intrauterine metabolic milieu rather than isolated glucose exposure. Altered placental signaling and metabolic modulation have been shown to influence fetal adiposity through complex endocrine pathways [16]. This mechanistic framework supports our observation that both GDM and elevated maternal BMI independently predicted LGA. The graded relationship between maternal glycemia and birth weight established in the HAPO study further strengthens this interpretation [17]. That landmark investigation demonstrated increasing rates of birth weight above the 90th percentile across the spectrum of maternal glucose levels. Our results parallel this continuum: the strong independent association between GDM and LGA reflects the biological plausibility of hyperglycemia-driven anabolic stimulation.

Maternal adiposity emerged as a critical amplifying factor in our cohort.

Stratification by treatment modality further clarified the impact of glycemic control. LGA was observed in 14.3% of diet-controlled GDM compared with 37.5% and 39.1% in OHA- and insulin-treated mothers respectively ($p=0.002$). These groups also demonstrated progressively higher mean fasting (88 vs 98 vs 104 mg/dL) and postprandial glucose levels (118 vs 142 vs 158 mg/dL). This gradient strongly suggests that severity of hyperglycemia, rather than treatment modality itself, determines the magnitude of fetal overgrowth.

Women with GDM had significantly higher pre-pregnancy BMI (**26.8 ± 3.9 vs 24.1 ± 3.2 kg/m²; $p = 0.001$**), and each unit increase in BMI independently increased LGA risk by **10% (aOR 1.10)**. Similar magnitudes of association have been reported in large Asian cohorts, where elevated pre-pregnancy BMI substantially increased LGA risk among women with GDM [18]. Comparable findings from Japan demonstrated that excessive pre-pregnancy BMI combined with gestational weight gain intensified LGA risk and operative delivery rates [19]. The consistency across populations suggests that maternal adiposity potentiates insulin resistance, enhances placental nutrient transfer, and augments fetal fat deposition. Familial predisposition also appeared relevant in our population. A positive family history of diabetes was significantly more frequent among GDM mothers (**52.0% vs 24.0%; $p = 0.001$**) and showed association with LGA in unadjusted analysis (**OR 1.98**). Prior research has shown that genetic predisposition interacts with BMI to increase metabolic vulnerability during pregnancy [20]. However, in our multivariate model, family history did not retain independent significance. This divergence suggests that inherited risk may operate primarily through the manifestation of maternal adiposity and active dysglycemia rather than directly influencing fetal size.

A similar pattern was observed with previous GDM. Although prior GDM increased the unadjusted odds of LGA nearly threefold in our cohort (**OR 2.80; $p = 0.032$**), it did not remain independently predictive. Larger population-based data have demonstrated increased recurrence and adverse perinatal outcomes in subsequent pregnancies [21]. The lack of independent association in our analysis likely reflects the dominant influence of current glycemic control over historical metabolic events. Recent work has also emphasized metabolic heterogeneity within GDM populations. Distinct insulin-resistant phenotypes have been linked to greater neonatal adiposity and adverse outcomes [22]. Our findings align with this concept: active metabolic expression—captured by current GDM status and maternal BMI—remained predictive, whereas background risk markers did not. Serial ultrasonographic surveillance provided additional insight. In our study, abdominal circumference did not differ significantly at 28 weeks; however, divergence became pronounced at 32 weeks (**29.4 vs 27.1 cm; $p < 0.001$**) and widened further at 36 weeks (**33.8 vs 31.2 cm; $p < 0.001$**). Estimated fetal weight followed a similar trajectory. These data suggest that sustained third-trimester hyperglycemia

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progressively drives adipose deposition rather than uniform skeletal growth.Growth parameters were comparable at 28 weeks (p=0.08), but significant divergence emerged after 32 weeks (p<0.001), indicating that prolonged third-trimester metabolic exposure plays a dominant role in accelerated fetal adiposity. This pattern is consistent with observations that abdominal adiposity correlates strongly with birth weight above the 90th percentile in diabetic pregnancies [23].

Although estimated fetal weight differed significantly between groups, only abdominal circumference retained independent predictive value (**aOR 1.22; p = 0.003**). Prior analyses have confirmed the utility of estimated fetal weight models in diet-controlled GDM [24]; however, our data suggest that AC may better reflect metabolically mediated fat accretion. This distinction is clinically relevant, as AC may serve as an earlier marker of disproportionate growth.

Maternal obesity alone has also been implicated in macrosomia, even in glucose-tolerant pregnancies [25]. This supports our finding that elevated BMI independently increased LGA risk, indicating that adiposity and glucose intolerance exert synergistic effects.

Efforts to refine prediction models have shown that combining maternal metabolic parameters with ultrasound markers improves diagnostic accuracy [26]. Our findings reinforce this multidimensional approach, as both maternal (BMI, GDM) and fetal (AC) variables independently contributed to LGA risk.

Early risk stratification may further improve outcomes. Predictive modelling of GDM prior to routine screening windows has been demonstrated in large cohorts [27]. Additionally, dietary patterns rich in refined carbohydrates have been shown to increase GDM risk [28], and maternal lipid profiles have been associated with LGA in diabetic pregnancies [29]. Although lipid parameters were not assessed in our study, the strong association between BMI and LGA suggests that excess substrate availability likely extends beyond glucose alone.

At the population level, regional variability in GDM prevalence has been linked to BMI distribution and socioeconomic transition [30]. Our findings from a South Indian tertiary center reflect this broader metabolic transformation and highlight its direct impact on fetal growth.

Limitations:

Despite the significant findings, certain limitations warrant consideration. First, although the calculated sample size was adequate to detect statistically meaningful differences in fetal growth parameters and LGA prevalence, the study was conducted at a single tertiary care center. This may limit external generalizability, particularly to rural populations or regions with differing dietary patterns, BMI distributions, or glycemic management protocols.Second, glycemic parameters were derived from routine clinical monitoring rather than standardized research-timed measurements. Variations in timing of fasting and postprandial sampling could introduce measurement variability, potentially influencing strength of associations observed between glycemic control and fetal growth outcomes.Third, while serial third-trimester assessments were performed, longitudinal fetal growth trajectories from early pregnancy were not evaluated. Early metabolic influences on placental programming and adiposity development therefore could not be assessed. Additionally, advanced sonographic markers of adiposity—such as fetal abdominal wall thickness, subcutaneous fat layers, or thigh soft tissue thickness—were not measured. Inclusion of these parameters might have enhanced characterization of disproportionate fat accretion.Fourth, maternal metabolic profiling was limited to glycemic indices. Important contributors to fetal overgrowth such as maternal lipid fractions, insulin levels, gestational weight gain patterns, and detailed dietary assessment were not analyzed. These unmeasured confounders may partially mediate the association between BMI, GDM, and LGA.Finally, as an observational case–control study, causal inferences must be interpreted cautiously. Although multivariate modelling adjusted for key variables, residual confounding cannot be entirely excluded.Future multicentric prospective cohort studies incorporating early pregnancy metabolic markers, lipid profiling, gestational weight gain trajectories, and advanced fetal adiposity imaging would provide deeper insight into mechanisms of metabolically driven fetal overgrowth.

Conclusion:

This study demonstrates a robust and clinically significant association between gestational diabetes mellitus and fetal overgrowth. LGA occurred in nearly one-third of GDM pregnancies, representing a three- to fourfold increased risk compared with normoglycemic pregnancies. Maternal pre-pregnancy BMI independently amplified this risk, while fetal abdominal circumference emerged as the strongest sonographic predictor of disproportionate growth. The pattern of growth acceleration observed predominantly in the third trimester supports the concept of sustained metabolic exposure driving preferential adipose deposition rather than uniform skeletal enlargement. Importantly, suboptimal glycemic control was associated with markedly higher LGA rates, underscoring that metabolic regulation—not merely diagnosis—determines fetal outcome.

These findings emphasize the need for a tiered preventive strategy: preconception weight optimization, early identification of high-risk women, stringent glycemic control throughout gestation, and focused third-trimester ultrasound surveillance—particularly abdominal circumference monitoring. Incorporating maternal BMI assessment and dynamic fetal abdominal growth tracking into routine obstetric protocols may improve risk stratification and guide timely obstetric decision-making. Effective metabolic control during pregnancy has the potential not only to reduce LGA prevalence but also to mitigate immediate perinatal complications and long-term intergenerational metabolic risk.

References:

1. Cunningham FG, Leveno KJ, Bloom SL, Dashe JS, Hoffman BL, Casey BM, et al. *Williams Obstetrics*. 26th ed. New York: McGraw Hill; 2022.
2. Rumack CM, Levine D, editors. *Diagnostic Ultrasound*. 5th ed. Philadelphia: Elsevier; 2018.
3. Kunarathnam V, Vadakekut ES, Mahdy H. Gestational Diabetes. [Updated 2025 Sep 15]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK545196/?
4. Mithal, Ambrish & Bansal, Beena & Kalra, Sanjay. (2015). Gestational diabetes in India: Science and society. Indian Journal of Endocrinology and Metabolism. 19. 10.4103/2230-8210.164031.
5. Mantri N, Goel AD, Patel M, Baskaran P, Dutta G, Gupta MK, Yadav V, Mittal M, Shekhar S, Bhardwaj P. National and regional prevalence of gestational diabetes mellitus in India: a systematic review and Meta-analysis. BMC Public Health. 2024 Feb 20;24(1):527. doi: 10.1186/s12889-024-18024-9. PMID: 38378536; PMCID: PMC10877914.
6. Zade SR, Anjana RM, Pradeepa R, Mohan V. Gestational diabetes mellitus in India: metabolic burden, challenges, and opportunities. *Explor Endocr Metab Dis*. 2025;2:101442.
7. Wang H, Li N, Chivese T, Werfalli M, Sun H, Yuen L, Hoegfeldt CA, Elise Powe C, Immanuel J, Karuranga S, Divakar H, Levitt N, Li C, Simmons D, Yang X; IDF Diabetes Atlas Committee Hyperglycaemia in Pregnancy Special Interest Group. IDF Diabetes Atlas: Estimation of Global and Regional Gestational Diabetes Mellitus Prevalence for 2021 by International Association of Diabetes in Pregnancy Study Group's Criteria. Diabetes Res Clin Pract. 2022 Jan;183:109050. doi: 10.1016/j.diabres.2021.109050. Epub 2021 Dec 6. PMID: 34883186.
8. Li Y, Ren X, He L, Li J, Zhang S, Chen W. Maternal age and the risk of gestational diabetes mellitus: A systematic review and meta-analysis of over 120 million participants. Diabetes Res Clin Pract. 2020 Apr;162:108044. doi: 10.1016/j.diabres.2020.108044. Epub 2020 Feb 1. PMID: 32017960.
9. Basu J, Datta C, Chowdhury S, Mandal D, Mondal NK, Ghosh A. Gestational Diabetes Mellitus in a Tertiary Care Hospital of Kolkata, India: Prevalence, Pathogenesis and Potential Disease Biomarkers. Exp Clin Endocrinol Diabetes. 2020 Apr;128(4):216-223. doi: 10.1055/a-0794-6057. Epub 2018 Dec 3. PMID: 30508848.

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ISSN: 1053-7899
Vol. 36 Issue 1, 2026, Pages: 3620-3626

10. Rabi T, Bandyopadhyay K, Adak SR. Frequency of gestational diabetes mellitus and associated risk factors amongst women attending antenatal clinic at a tertiary care hospital of West Bengal, India. *Int J Reprod Contracept Obstet Gynecol*. 2023;12(6):1706–1709.
11. Gunjan, & Khare, Indu & Kumar, Ashutosh & Sinha, Anjana. (2024). Examination of gestational diabetes mellitus at a tertiary care hospital. International Journal of Research in Medical Sciences. 12. 3279-3283. 10.18203/2320-6012.ijrms20242296.
12. Bahl S, Dhabhai N, Taneja S, Mittal P, Dewan R, Kaur J, Chaudhary R, Bhandari N, Chowdhury R. Burden, risk factors and outcomes associated with gestational diabetes in a population-based cohort of pregnant women from North India. BMC Pregnancy Childbirth. 2022 Jan 14;22(1):32. doi: 10.1186/s12884-022-04389-5. PMID: 35031013; PMCID: PMC8759176.
13. Ruge TC, Kanchana N. Prevalence of gestational diabetes mellitus using IADPSG and DIPSI criteria: a cross-sectional study. *Int J Reprod Contracept Obstet Gynecol*. 2020;9(6):2408–2414
14. Chakraborty A, Yadav S. Prevalence and determinants of gestational diabetes mellitus among pregnant women in India: an analysis of National Family Health Survey Data. BMC Womens Health. 2024 Feb 29;24(1):147. doi: 10.1186/s12905-024-02936-0. PMID: 38424617; PMCID: PMC10902981.
15. Batra, Niti & Ahirwar, Mohini & Chaurasia, Karishma & Sirpurkar, Manik. (2025). Incidence of gestational diabetes mellitus among Indian women. Bioinformation. 21. 2763-2766. 10.6026/973206300212763.
16. Rolfo A, Nuzzo AM, De Amicis R, Moretti L, Bertoli S, Leone A. Fetal-Maternal Exposure to Endocrine Disruptors: Correlation with Diet Intake and Pregnancy Outcomes. Nutrients. 2020 Jun 11;12(6):1744. doi: 10.3390/nu12061744. PMID: 32545151; PMCID: PMC7353272.
17. HAPO Study Cooperative Research Group; Metzger BE, Lowe LP, Dyer AR, Trimble ER, Chaovarindr U, Coustan DR, Hadden DR, McCance DR, Hod M, McIntyre HD, Oats JJ, Persson B, Rogers MS, Sacks DA. Hyperglycemia and adverse pregnancy outcomes. N Engl J Med. 2008 May 8;358(19):1991-2002. doi: 10.1056/NEJMoa0707943. PMID: 18463375.
18. Lin L, Wu J, Xu L, Fang J, Lin J. Maternal body mass index and risk of fetal overgrowth in women with gestational diabetes Mellitus in Southeast China: a retrospective cohort study. Diabetol Metab Syndr. 2023 Jun 8;15(1):121. doi: 10.1186/s13098-023-01093-y. PMID: 37291681; PMCID: PMC10249232.
19. Saito Y, Kobayashi S, Ikeda-Araki A, Ito S, Miyashita C, Kimura T, Hirata T, Tamakoshi A, Mayama M, Noshiro K, Nakagawa K, Umazume T, Chiba K, Kawaguchi S, Morikawa M, Cho K, Watari H, Ito Y, Saijo Y, Kishi R; Japan Environment and Children's Study (JECS) group. Association between pre-pregnancy body mass index and gestational weight gain and perinatal outcomes in pregnant women diagnosed with gestational diabetes mellitus: The Japan Environment and Children's Study. J Diabetes Investig. 2022 May;13(5):889-899. doi: 10.1111/jdi.13723. Epub 2021 Dec 22. PMID: 34845867; PMCID: PMC9077720.
20. Lewandowska M. Gestational Diabetes Mellitus (GDM) Risk for Declared Family History of Diabetes, in Combination with BMI Categories. Int J Environ Res Public Health. 2021 Jun 28;18(13):6936. doi: 10.3390/ijerph18136936. PMID: 34203509; PMCID: PMC8293805.
21. Kim MJ, Cho GJ, Bae JG, Lee GS, Wie JH, Park S, Seong WJ, Ko HS. Analysis of risk factors for the recurrence of gestational diabetes in subsequent pregnancy: A nationwide population-based study in South Korea. Medicine (Baltimore). 2025 Aug 22;104(34):e44044. doi: 10.1097/MD.0000000000044044. PMID: 40859514; PMCID: PMC12385086.
22. Immanuel J, Simmons D, Harreiter J, Desoye G, Corcoy R, Adelantado JM, Devlieger R, Lapolla A, Dalfra MG, Bertolotto A, Wender-Ozegowska E, Zawiejska A, Dunne FP, Damm P, Mathiesen ER, Jensen DM, Andersen LLT, Hill DJ, Jelsma JGM, Kautzky-Willer A, Galjaard S, Snoek FJ, van Poppel MNM. Metabolic phenotypes of early gestational diabetes mellitus and their association with adverse pregnancy outcomes. Diabet Med. 2021 Feb;38(2):e14413. doi: 10.1111/dme.14413. Epub 2020 Oct 14. PMID: 32991758.
23. Rauh M, Voigt M, Kappelmeyer M, Schmidt B, Köninger A. Correlation of sonographically measured fetal abdominal wall thickness with birth weight in diabetes. Eur J Obstet Gynecol Reprod Biol. 2024 Dec;303:9-14. doi: 10.1016/j.ejogrb.2024.10.003. Epub 2024 Oct 4. PMID: 39395246.
24. Husslein H, Worda C, Leipold H, Szalay S. Accuracy of Fetal Weight Estimation in Women with Diet Controlled Gestational Diabetes. Geburtshilfe Frauenheilkd. 2012 Feb;72(2):144-148. doi: 10.1055/s-0031-1298278. PMID: 25284831; PMCID: PMC4168337.
25. Owens LA, O'Sullivan EP, Kirwan B, Avalos G, Gaffney G, Dunne F; ATLANTIC DIP Collaborators. ATLANTIC DIP: the impact of obesity on pregnancy outcome in glucose-tolerant women. Diabetes Care. 2010 Mar;33(3):577-9. doi: 10.2337/dc09-0911. Epub 2010 Jan 12. PMID: 20067952; PMCID: PMC2827510.
26. Zhang J, Wu X, Song Q. Analytical Comparison of Risk Prediction Models for the Onset of Macrosomia Based on Three Statistical Methods. Dis Markers. 2022 Sep 10;2022:9073043. doi: 10.1155/2022/9073043. PMID: 36124028; PMCID: PMC9482546.
27. Zheng T, Ye W, Wang X, Li X, Zhang J, Little J, Zhou L, Zhang L. A simple model to predict risk of gestational diabetes mellitus from 8 to 20 weeks of gestation in Chinese women. BMC Pregnancy Childbirth. 2019 Jul 19;19(1):252. doi: 10.1186/s12884-019-2374-8. PMID: 31324151; PMCID: PMC6642502.
28. He JR, Yuan MY, Chen NN, Lu JH, Hu CY, Mai WB, Zhang RF, Pan YH, Qiu L, Wu YF, Xiao WQ, Liu Y, Xia HM, Qiu X. Maternal dietary patterns and gestational diabetes mellitus: a large prospective cohort study in China. Br J Nutr. 2015 Apr 28;113(8):1292-300. doi: 10.1017/S0007114515000707. Epub 2015 Mar 30. PMID: 25821944.
29. Gutaj P, Wender-Ożegowska E, Brązert J. Maternal lipids associated with large-for-gestational-age birth weight in women with type 1 diabetes: results from a prospective single-center study. Arch Med Sci. 2017 Jun;13(4):753-759. doi: 10.5114/aoms.2016.58619. Epub 2016 Mar 16. PMID: 28721142; PMCID: PMC5510499.
30. Chakraborty A, Yadav S. Prevalence and determinants of gestational diabetes mellitus among pregnant women in India: an analysis of National Family Health Survey Data. BMC Womens Health. 2024 Feb 29;24(1):147. doi: 10.1186/s12905-024-02936-0. PMID: 38424617; PMCID: PMC10902981.
31. Schaefer-Graf UM, Kjos SL, Kilavuz O, Plagemann A, Brauer M, Dudenhausen JW, Vetter K. Determinants of fetal growth at different periods of pregnancies complicated by gestational diabetes mellitus or impaired glucose tolerance. Diabetes Care. 2003 Jan;26(1):193-8. doi: 10.2337/diacare.26.1.193. Erratum in: Diabetes Care. 2003 Apr;26(4):1329. PMID: 12502680.
32. Li M, Hinkle SN, Grantz KL, Kim S, Grewal J, Grobman WA, Skupski DW, Newman RB, Chien EK, Sciscione A, Zork N, Wing DA, Nageotte M, Tekola-Ayele F, Louis GMB, Albert PS, Zhang C. Glycaemic status during pregnancy and longitudinal measures of fetal growth in a multi-racial US population: a prospective cohort study. Lancet Diabetes Endocrinol. 2020 Apr;8(4):292-300. doi: 10.1016/S2213-8587(20)30024-3. Epub 2020 Mar 2. PMID: 32135135; PMCID: PMC7676113.
33. Catalano PM, Thomas AJ, Huston LP, Fung CM. Effect of maternal metabolism on fetal growth and body composition. Diabetes Care. 1998 Aug;21 Suppl 2:B85-90. PMID: 9704233.