

Research Article

Phenotypic Variations and Birth Weight Trends in Neonates of Gestational Diabetic Mothers- Hospital Based Cohort Study

Dr. Pramod Padasalimani^{1*}, Dr. Gr Basanthkumar², Dr. Savitri Honakeri³

^{1*}Senior resident, Department of paediatrics, koppal institute of medical science koppal.

²Professor, Department of paediatrics, jjm medical college davangere.

³Assistant professor, Department of paediatrics surgery, kmcri hubballi.

Corresponding Author: Dr Pramod Padasalimani Senior resident, Department of paediatrics, koppal institute of medical science koppal.

Email: pramodpadasali@gmail.com

Received: 01.06.26, Revised: 04.07.26, Accepted: 03.08.26

ABSTRACT

Background: Gestational diabetes mellitus (GDM) represents a significant metabolic challenge during pregnancy, directly impacting fetal growth and development. While the classical phenotype of the infant of a diabetic mother is large for gestational age (LGA), birth weight alone may not capture the full extent of metabolic alterations.

Methods: A hospital-based cohort study was conducted on 105 neonates born to GDM mothers admitted to a neonatal intensive care unit (NICU) to assess phenotypic categories and immediate clinical outcomes. These findings were comparatively analysed against recent literature to identify changing trends in neonatal assessment.

Results: In the current clinical cohort, infants exhibited varied phenotypes: 33.3% were LGA, 53.3% were average for gestational age (AGA), and 13.3% were small for gestational age (SGA). High rates of short-term morbidities were observed, notably hypoglycaemia (64.8%) and respiratory distress syndrome (50.5%).

Conclusion: The phenotypic presentation of infants of GDM mothers is a spectrum ranging from growth restriction to macrosomia. Recent trends indicate a paradigm shift from relying solely on standard birth weight percentiles toward direct body composition analysis to better predict neonatal complications.

INTRODUCTION

Maternal diabetes mellitus is the major cause of large-for-gestational-age (LGA) infants. The excessive growth generally results from the anabolic effects of high fetal insulin levels produced in response to excessive maternal blood glucose during gestation. Consequently, the phenotype of the infant of the diabetic mother is traditionally perceived as being macrocosmic.(1-4) In the first half of pregnancy, the fetus is exposed primarily to hyperglycaemia, which, without secondary hyperinsulinemia, results in slowing of fetal growth. During the second half of pregnancy, hypertrophic islet cells respond to hyperglycemia with an increase in insulin production. This potent combination of hyperinsulinemia, a major anabolic hormone, and hyperglycemia, a major anabolic fuel, results in a cascade of third-trimester events that culminate in a striking increase in fat stores and a modest 12% increase in protein stores. Head circumferences are not typically increased.(5-11) In the past, up to 60% of IDMs have been reported to be macrosomic, as

defined by a weight more than the ninety-fifth percentile or weight >4000g, although this rate has declined to 20% to 35% , most likely secondary to aggressive diagnosis and treatment of diabetes during pregnancy. Macrosomia places the IDM at greater risk for birth trauma because of cephalopelvic disproportion. Appropriate evaluation before delivery includes a review of maternal glycemic control, estimated fetal weight and size, and bio-physical profile assessments.(6-10) Not surprisingly, caesarean delivery rates remain higher in IDMs with fetal macrosomia documented on prenatal ultrasound. Diabetic women with additional risk factors for macrosomia, such as obesity and post-maturity, have the highest risk of delivering a macrosomic fetus and should be monitored closely for this occurrence. A small (<5%) subgroup of fetuses, usually carried by mothers with advanced diabetic vascular disease, is at risk for fetal growth deceleration, defined as birth weight less than the fifth percentile for gestational age. The placental

vascular insufficiency frequently is accompanied by maternal hypertension and results in protein-energy malnutrition (manifested as fetal growth deceleration) and restricted oxygen delivery (manifested as secondary polycythemia). Their tenuous fetal physiology places them at increased risk of being compromised at delivery. These pathologic conditions may be independent of maternal glycemic control. As with the macrosomic fetus, appropriate evaluation includes biophysical profile assessment and estimation of fetal weight. Depending on complex gene-environment interactions, the phenotypic presentation can vary. Because insulin acts as a primary stimulus for growth in the fetus, any defect in fetal insulin secretion can result in decreased fetal growth and possible growth restriction, the infant's phenotype can range from intrauterine growth restriction, through normal fetal growth to macrosomia and in instances of genetic imprinting, such as Beckwith-Wiedemann syndrome, can cause an offspring to present with the exact macrosomic phenotype of a GDM infant even if the mother has normal glucose tolerance.(11-18) the definition of macrosomia, i.e., excessive body size, has not changed much in the past decades. In reference to fetal growth, macrosomia is commonly defined as either birth weight greater than the 90th centile for gestational age or 4,000 g, independent of gestational age or sex. However, both of these definitions fail to take into account other factors that may be significant variables relating to fetal growth, such as sex, socioeconomic status, ethnicity, parity, and geographic variables such as altitude. We are all aware of the recent trends for increases in the prevalence of adult overweight and obesity. However, there have also been significant increases in overweight in children, defined as 95th centile of BMI for age over the last decade, particularly in minority groups. In children as young as 2–5 years old, the prevalence of obesity has increased from 5 to 10.4% over the last 15–20 years (18-22) (23). In Denmark from 1990 through 1999, the percent of babies weighing 4,000g at birth has increased from 16.7 to 20% (23). In Sweden, there has been

a 23% increase in birth weight of large-for-gestational-age (LGA) babies, defined as 2 SDs birth weight for gestational age, over the same time period (23). In North America, although average birth weights have increased only modestly, the percent of term small-for-gestational-age (SGA) babies (both white and black) has decreased in the U.S. by 11–12%, whereas in Canada, there has been a 27% decrease in SGA babies over the period from 1985 to 1998. In contrast, the percent LGA babies has increased during the same time period in the U.S. by 5% (white) and 9% (black) and by 24% in Canada. Hence, not only are the adult and adolescent populations experiencing an increase in the prevalence of obesity, but the same may be occurring at the time of birth.(23) This article evaluates the weight distribution and clinical phenotypes of infants of gestational diabetic mothers in a clinical cohort and compare these findings with recent studies to highlight current diagnostic trends.

METHODOLOGY

A hospital-based cohort study was conducted observing 105 newborn babies delivered to diabetic mothers and admitted to the NICU in hospital attached to JJM Medical College, Davangere from March 2021 to October 2022. Neonates were categorized based on birth weight for gestational age into SGA, AGA, and LGA cohorts, and managed complications. Data entered into Microsoft Excel and was analysed using SPSS 22 version software. A total of 105 newborns were observed according to inclusion and exclusion criteria. Data Collection Technique According to inclusion criteria. A structural questionnaire was filled up for each IDM after taking informed written consent from their guardian. Detail socioeconomic, maternal medical and diabetic status, drug, obstetrical and neonatal birth history were recorded. Infant's weight, length, ponderel index was measured and documented as per standard Fenton's charts and routine blood test were estimated in the laboratory of medical college and were treated as per standard hospital protocol.

RESULTS

Maternal Demographics and Characteristics

		Frequency	Percentage
Gestational Age	< 37 Weeks	20	19%
	>37 Weeks	85	81%

Subjects (N=105) Frequency (N) Percentage (%)

Subjects (N=105)	Mean	SD	Median	Minimum	Maximum
GA (In Weeks)	37.54	1.80	38.00	30.00	40.00

In the study, majority of the mothers had crossed 37 weeks of gestation (81.0%), while remaining were less than 37 weeks of gestation

(19.0%). The mean gestational age of the mothers was 37.54 ± 1.80 weeks

Distribution of Mothers by Gestational Age

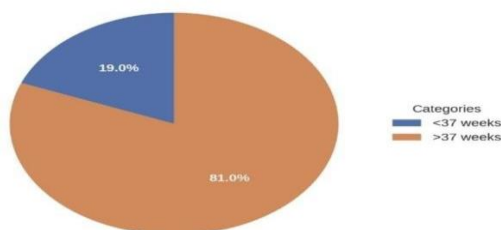


Figure 1 – Distribution of Mothers Based On Gestational Age

		Frequency	Percentage
Mode Of Delivery	NVD	46	43.8%
	Assisted Delivery	8	7.6%
	LSCS	51	48.6%

In the study, the most common mode of delivery was LSCS (48.6%). The next majority

was normal vaginal delivery (43.8%), followed by assisted delivery (7.6%).

Distribution of Mothers by Mode of Delivery

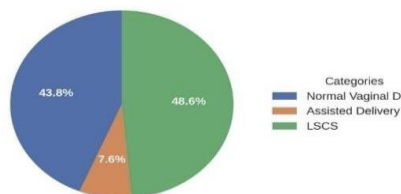


Figure 2: Distribution of the Mothers Based On the Mode of Delivery

Parity	Frequency	Percentage
Primi	50	47.6
Multi	55	52.4

Obstetric SCORE	Mean	SD	Median	Minimum	Maximum
	1.87	1.01	2.00	1.00	6.0

In the study, majority of the mothers were multigravida (52.4%), while remaining were

primigravida (47.6%). The mean obstetric score among the mothers was 1.87 ± 1.01 .

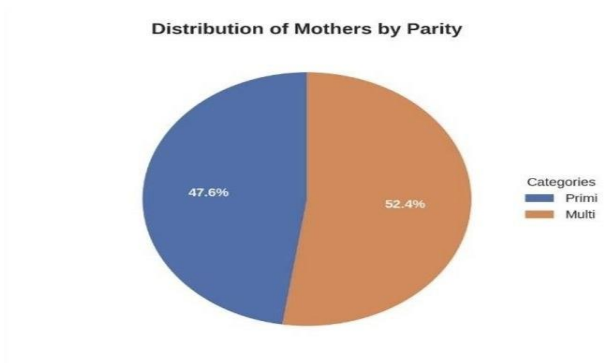


Figure 3: Distribution of the Mothers Based On Parity

	N= 105	Frequency	Percentage
Duration of GDM	Early Onset	20	19%
	Late Onset	85	81%

In the study, majority of the mothers developed GDM at late onset (81.0%), while remaining was early in onset GDM (19.0%).

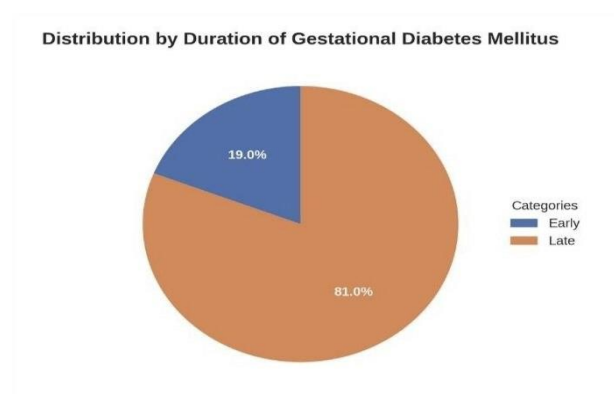


Figure 4: Distribution of the Mothers Based On the Duration of Gestational Diabetes Mellitus

		Frequency	Percentage
Gender	MALE	63	60%
	FEMALE	42	40%

In the study, majority of the neonates were males (60.0%), and the remaining were females (40.0%).

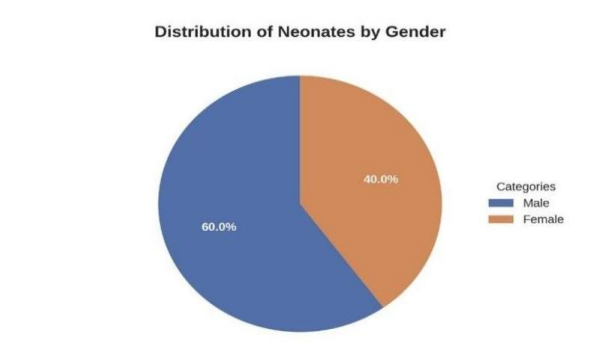


Figure 5: Distribution of the Neonates Based On Gender

	N-105	Frequency	Percentage
Birth Weight	<1.99kg	14	13.3
	2.00-2.49kg	6	5.7
	2.50-2.99kg	29	27.6
	3.00-3.49kg	25	23.8
	3.50-3.99 Kg	27	25.7
	>4.00 Kg	4	3.8

5 In the study, majority of the neonates weighed between 2.50 and 2.99 kg (27.6%). The next majority was 3.50 to 3.99 kg (25.7%),

followed by 3.00 to 3.49 kg (23.8%). The mean birth weight of the neonates was 2.97 ± 0.67 kg.

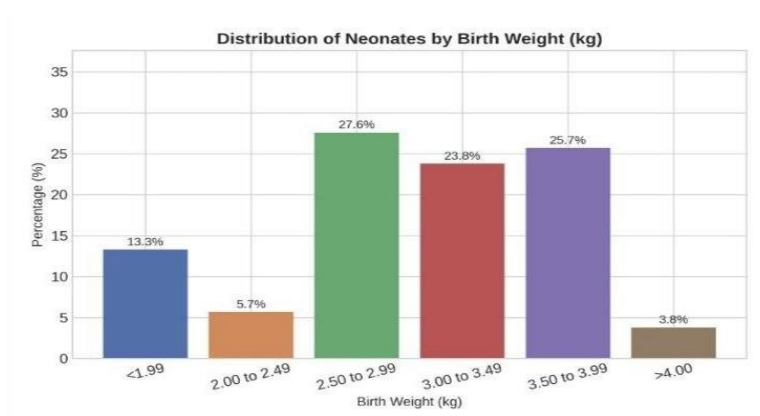


Figure 6: Distribution of the Neonates Based On Birth Weight Subjects

		Frequency	Percentage
Growth	Small For GA	14	13.3
	Appropriate For GA	56	53.33
	Large For GA	35	33.3

In the study, majority of the neonates were found to be in the range of appropriate weight for gestation (53.3%). The next majority were

large for GA (33.3%), followed by small for GA (13.3%).

Distribution of Neonates by Weight for Gestation

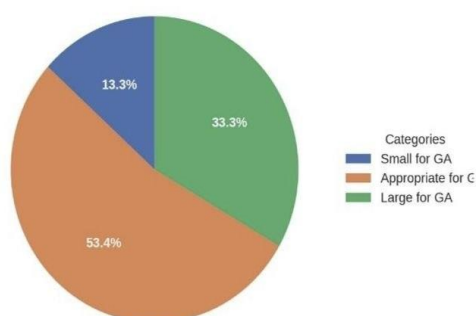


Figure 7: Distribution of the neonates based on the weight for gestation
Associations between Maternal and Neonatal Factors

Subject (n=105)		Onset of GDM				P value
		Early onset		Late onset		
		N	%	N	%	
Birth weight	<1.99kg	4	20.0	10	11.8	0.077
	2.0-2.49kg	3	15.0	3	3.5	
	2.50-2.99kg	4	20.0	25	29.4	
	3.00-3.49kg	5	25.0	20	23.5	
	3.50-3.99kg	2	10.0	25	29.4	
	>4.00kg	2	10.0	2	2.4	
Growth	SGA	3	15.0	11	12.9	0.368
	AGA	13	65.0	43	50.6	
	LGA	4	20.0	31	36.5	

In the study, majority of the neonates born to the mothers with early onset GDM weighed below 3.00 kg. On other hand, among those with late onset GDM, their neonates weighed above 3.00 kg. However, in both the groups, majority of the neonates were appropriate for GA. Thus on analysing the association between neonatal growth and duration of GDM, the study found no significant relation, thereby suggesting that the onset of GDM did not significantly affect the birth weight and growth of the neonates as per gestation in present study

In the current study cohort (n=105), the mean birth weight of the infants was 2.97 kg. Phenotypic assessment revealed a wide variance in fetal growth:AGA (Average for Gestational Age): 53.3% LGA (Large for Gestational Age): 33.3% SGA (Small for Gestational Age): 13.3% Metabolic complications were highly prevalent in this NICU cohort, with hypoglycemia developing in 64.8% of the neonates. Respiratory Distress Syndrome (RDS) was observed in 50.5% of cases and they were managed as per protocol

DISCUSSION

To contextualize the current study's findings, they compared with recent, large-scale literature examining GDM offspring. A 2025 comparative study investigating the impact of GDM on neonatal birth weight found that neonates of GDM mothers had significantly higher birth weights overall (mean 3689 g) compared to non-GDM controls (mean 3143 g). In this recent study, the macrosomia rate (birth weight ≥ 4000 g) was 25.5%, and the SGA rate was 8.5%. The LGA rate in our current study (33.3%) is notably higher than the 25.5% observed in the 2025 comparative cohort. Similarly, the current study recorded a higher incidence of SGA infants (13.3% vs. 8.5%) (18-

23). this wider phenotypic variance likely reflects the high-risk nature of a strictly NICU-admitted population. The current study noted a 64.8% incidence of hypoglycemia. In contrast, the 2025 study reported a neonatal hypoglycemia rate of only 17% in GDM cases, alongside a NICU admission rate of 20.2%. The elevated morbidity in the current cohort shows the critical vulnerability of infant of GDM requiring intensive care interventions.

In recent days, the trend in perinatal research has shifted away from evaluating birth weight alone. Neonatal body composition may be a sensitive marker of metabolic effects on the fetus caused by suboptimal glycemic control of mother during pregnancy. Current literature demonstrates that even when controlling for overall birth weight, infants born to maternal GDM have significantly different body compositions compared to healthy babies.

In infant of GDM, anthropometric parameters, fat mass, or body fat percentage did not differ between infants with or without postnatal hypoglycemia. This suggests that while fetal hyperinsulinemia causes excess fat deposition (macrosomia), the acute drop in blood glucose after cord clamping is by mechanisms partially independent of the infant's absolute fat mass. By focusing strictly on NICU admissions, the study provides evaluation of immediate postnatal complications.

The study actively categorized infants into LGA, AGA, and SGA groups instead of taking uniform macrosomia, which shows that the GDM phenotype is a broad spectrum. But being limited to a NICU environment, the study represents a higher-risk population, which may give false high complication rates compared to well-babies and not measuring body adiposity or fat-free mass, which is a growing limitation in modern neonatal metabolic assessments. Non availability of prepregnancy BMI and

glycemic control is one more drawback of study which are critical independent predictors of neonatal macrosomia (23)

CONCLUSION

The phenotypic presentation of the infant of a diabetic mother is complex and have various presentation. While macrosomia and the LGA phenotype remain classic hallmark which affecting nearly a 1/3rd of affected pregnancies—the clinical study includes significant populations of AGA and SGA infants. High-acuity NICU populations demonstrate drastically increased rates of acute metabolic decompensation, particularly hypoglycemia. In summary, the infant of a GDM mother may have a variable phenotype based on the interaction of genes and the in utero environment. And, the macrosomic fetus who presents like the infant of a GDM mother may have the possibility of other genetic or metabolic dysfunctions mimicking GDM. Birth weight alone may not be a sensitive enough measure of fetal growth to assess the effects of GDM. Importance should be given to estimation of fetal adiposity, Ponderal Index (weight/length³). To better understand the long-term metabolic programming of these vulnerable infants.

REFERENCE

1. Jansen C, Greenspoon SJ, Palmer MS, Diabetes Mellitus & Pregnancy. Current obstetric & gynaecologic diagnosis & treatment; 9th Ed. 2003; 18: 317-321
2. Avery's Diseases of the newborn, 2005; 8th Ed: 71-86
3. Sudarshan K, Jain S, Jain RK. Study of morbidity and mortality pattern in infants born to diabetic mothers. J Obstet Gynaecol India. 1987;37:481-.
4. Weintrob N, Karp M, Hod M. Short-and long-range complications in offspring of diabetic mothers. Journal of Diabetes and its Complications. 1996 Sep 1;10(5):294-301.
5. Catalano PM, Thomas A, Huston-Presley L, Amini SB: Increased fetal adiposity: a very sensitive marker of abnormal in utero development. Am J Obstet Gynecol 189:1698-1704, 2003
6. Durnwald C, Huston-Presley L, Amini S, Catalano P: Evaluation of body composition of large-for-gestational-age infants of women with gestational diabetes mellitus compared with women with normal glucose tolerance levels. Am J Obstet Gynecol 191:804-808, 2004
7. Uvena-Celebrezze J, Fung C, Thomas AJ, Hoty A, Huston-Presley L, Amini SB, Catalano PM: Relationship of neonatal body composition to maternal glucose control in women with gestational diabetes mellitus. J Matern Fetal Neonatal Med 12:396-401, 2002
8. Jovanovic-Peterson L, Peterson CM, Reed GF, Metzger BE, Mills JL, Knopp RH, Aarons JH: Maternal post prandial glucose levels and infant birth weight: The Diabetes in Early Pregnancy Study. Am J Obstet Gynecol 164:103-111, 1991
9. Langer O, Rodriguez DA, Xenaris EM, McFarlane MB, Berkus MD, Arrendondo F: Intensified versus conventional management of gestational diabetes. Am J Obstet Gynecol 170:1036-1047, 1994
10. BuchananTA, KjosSL, ManaoroMN, Wu P, Madrilejo NG, Gonzalez M, Nunez V, Pantoja PM, Xiang A: Use of fetal ultrasound to select metabolic therapy for pregnancies complicated by mild gestational diabetes. Diabetes Care 17: 275-283, 1994
11. WeissP, HofmannH: Diagnosis and treatment of gestational diabetes according to amniotic fluid insulin levels. Arch Gynecol 239:81-91, 1986
12. Freinkel N: The Banting Lecture 1980: Of pregnancy and progeny. Diabetes 29: 1023, 1980
13. Knoop RH, Bergelin RO, Wahl PW, Walden CE: Relationships of infant birth size to maternal lipoproteins, apoproteins, fuels, hormones, clinical chemistries, and body weight at 36 weeks gestation. Diabetes 34 (Suppl. 2):71-77, 1985
14. Schaeffer-Graf UM, Kjos SL, Kilaouz 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 26:193-198, 2003
15. Langer O, Yogev Y, Xenakis EMJ, Brustman L: Overweight and obese in gestational diabetes: the impact on pregnancy outcome. Am J Obstet Gynecol 192:1368-1376, 2005
16. Catalano PM, Ehrenberg HM: The short and long-term implications of maternal obesity on the mother and her offspring. BJOG 113:1126-33, 2006
17. Stoll JB. Infants of diabetic mothers,

- Kliegman: Nelson Textbook of Paediatrics, 2022: 20th ed. Pg. 107.
19. Gabbe SG. Pregnancy in women with diabetes mellitus: The beginning. Clinics in perinatology. 1993 Sep 1;20(3):507-15.
 20. Hussain M, Irshad M, Khattak AK, Khan B. Frequency of various neonatal complications in infants born to diabetic mothers-a hospital base study. Journal of Postgraduate Medical Institute. 2011;25(3):227-32.
 21. Shirazi H, Riaz S, Mahmood I and Gul S. Neonatal outcome of diabetic pregnancy. Ann. Pak. Inst. Med. Sci. 2010;6(2):107-12.
 22. Prabhavathi R, Basavaraj PK, Udaykumar S. A study of perinatal and outcome in infants born to diabetic mothers. Int J Adv Med. 2015;2:246-9.
 23. Anjum SK, Yashodha HT. A study of neonatal outcome in infants born to diabetic mothers at a tertiary care hospital. Int J Contemp Pediatr. 2018 Mar;5(2):489-92.
 24. Tyralla EE. The infant of the diabetic mother. Obstetrics and Gynecology Clinics. 1996 Mar 1;23(1):221-41.
 25. Catalano PM, Thomas A, Huston-Presley L, Amini SB. Phenotype of infants of mothers with gestational diabetes. Diabetes Care. 2007 Jul;30(Suppl 2):S156-S160. doi:10.2337/dc07-s209.