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Research Article

Cardiovascular Abnormalities in Newborns of Gestational Diabetic Mothers

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Abstract
Background: Maternal diabetes mellitus, including gestational diabetes mellitus (GDM), poses significant risks to fetal heart development. The teratogenic impact of maternal hyperglycemia during organogenesis, combined with fetal hyperinsulinemia in late gestation, leads to structural congenital heart defects (CHD) and functional myocardial alterations.
Objective: To evaluate the spectrum of cardiovascular abnormalities in neonates born to gestational diabetic mothers admitted to a tertiary care neonatal intensive care unit (NICU) and analyze their correlation with maternal GDM onset and neonatal parameters.
Methods: A prospective observational study was conducted at the Neonatal Intensive Care Unit (NICU), Department of Pediatrics, J. J. Medical College, Davangere. A total of 105 neonates born to mothers diagnosed with gestational diabetes mellitus were enrolled. Maternal parameters (gestational age, mode of delivery, GDM onset) and neonatal variables (birth weight, weight for gestational age, APGAR scores) were recorded. Cardiovascular evaluation was performed using clinical examination and 2D Echocardiography. Statistical analysis was carried out using Chi-square test, Fisher's exact test, and independent t-test ($p < 0.05$ considered significant).
Results: Cardiovascular abnormalities were identified in 9.5% (n = 10) of neonates. Patent Ductus Arteriosus (PDA) associated with moderate-to-severe Pulmonary Arterial Hypertension (PAH) was the most frequent cardiovascular finding (7.6%, n = 8), followed by Asymmetric Septal Hypertrophy (1.9%, n = 2), Coarctation of the Aorta (1.0%, n = 1), and antenatal Pericardial Effusion (1.0%, n = 1). Early onset of maternal GDM was associated with a statistically significant increase in adverse neonatal mortality and severe cardiovascular compromise compared to late-onset GDM ($p = 0.003$).
Conclusion: Maternal gestational diabetes is strongly associated with structural and functional cardiovascular abnormalities in newborns, predominantly PDA with PAH and hypertrophic cardiomyopathy. Early maternal screening, strict glycemic control, universal fetal echocardiography, and routine postnatal cardiovascular evaluation are essential to minimize neonatal cardiovascular morbidity and mortality.

Keywords: Infant of Diabetic Mother, Gestational Diabetes Mellitus, Cardiovascular Abnormalities, Patent Ductus Arteriosus, Asymmetric Septal Hypertrophy, Neonatal Echocardiography, Pulmonary Arterial Hypertension.

INTRODUCTION

Diabetes complicating pregnancy, whether pregestational or gestational diabetes mellitus (GDM), poses a major clinical challenge in perinatal medicine. Gestational diabetes mellitus arises during pregnancy and reflects varying degrees of carbohydrate intolerance. Cardiovascular malformations remain among the most severe structural and functional complications observed in infants of diabetic mothers (IDMs). The incidence of congenital heart disease (CHD) in diabetic pregnancies ranges between 3% and 9%, representing a risk 2.5 to 10 times higher than that of the general obstetric population.

The pathophysiology linking maternal hyperglycemia to fetal cardiovascular derangements is complex and multifactorial. According to the Pedersen hypothesis, maternal hyperglycemia leads to direct transfer of excessive glucose across the placenta, stimulating fetal pancreatic beta-cells to produce excess insulin. During early embryogenesis (prior to 7 weeks of gestation),

maternal hyperglycemia exerts direct teratogenic effects during critical periods of cardiac organogenesis, resulting in cardiac looping defects, altered left-right patterning, and septal malformations. In later gestation, hyperinsulinemia stimulates insulin receptors located within the fetal myocardium, causing excessive intracellular glycogen deposition and cardiac cellular hypertrophy. This hyperinsulinemic environment drives the development of hypertrophic cardiomyopathy, classically presenting as asymmetric septal hypertrophy (ASH), which can cause dynamic subaortic outflow tract obstruction.

In addition to structural malformations and myocardial hypertrophy, infants of diabetic mothers are prone to hemodynamically significant lesions, such as persistent Patent Ductus Arteriosus (PDA) accompanied by elevated pulmonary vascular resistance leading to Pulmonary Arterial Hypertension (PAH), Coarctation of the Aorta, and fetal/neonatal pericardial effusions.

. This study was undertaken to evaluate the spectrum of cardiovascular abnormalities in infants born to gestational diabetic mothers in a tertiary care center and to correlate these cardiac findings with maternal and neonatal parameters.

MATERIALS AND METHODS

Study Design and Setting

This prospective observational study was conducted in the Neonatal Intensive Care Unit (NICU) attached to the Department of Pediatrics, J. J .m. Medical College (JJMMC), Davangere, Karnataka, India.

Study Population

The study included 105 consecutive neonates born to mothers diagnosed with Gestational Diabetes Mellitus (GDM) who were admitted to the NICU during the study period.

Eligibility Criteria

- **Inclusion Criteria:** All live-born neonates of mothers diagnosed with Gestational Diabetes Mellitus according to standard diagnostic criteria (WHO / DIPSI criteria) admitted to the NICU.
- **Exclusion Criteria:** Neonates with major non-cardiac genetic or chromosomal syndromes, or cases where parental informed consent was not obtained.

Data Collection and Cardiovascular Evaluation

Detailed maternal clinical history was recorded using a standardized proforma, capturing parameters such as maternal gestational age, parity, mode of delivery (Lower Segment Caesarean Section [LSCS], normal vaginal delivery, or assisted delivery), duration/onset of GDM (categorized as early-onset vs. late-onset GDM), and relevant antenatal events.

Neonatal demographic and clinical variables were documented at admission, including gender, birth weight, weight for gestational age (Small for Gestational Age [SGA], Appropriate for Gestational Age [AGA], Large for Gestational Age [LGA]), and APGAR scores at 1 and 5 minutes.

Comprehensive cardiovascular assessment was conducted for all subjects, including detailed clinical examination (assessment of murmurs, femoral pulses, pre- and post-ductal oxygen saturation, precordial activity), chest radiography, and 2D Echocardiography with Color Doppler. Echocardiographic evaluations focused on identifying:

1. Structural heart defects (e.g., Patent Ductus Arteriosus, Coarctation of the Aorta, septal defects).
2. Functional myocardial abnormalities, specifically Asymmetric Septal Hypertrophy (interventricular septal thickness in diastole).
3. Hemodynamic alterations, including Pulmonary Arterial Hypertension (PAH) and Pericardial Effusion.

Statistical Analysis

Data were compiled and analyzed using SPSS version 22.0 (IBM SPSS Statistics, Armonk, NY, USA) and MS Excel. Categorical variables were presented as absolute frequencies and percentages. Continuous variables were expressed as mean ± standard deviation (SD) or median where applicable. The Chi-square test was utilized to analyze categorical comparisons. Fisher's exact test was applied for 2×2 tables where cell counts were below expected values. Independent t-tests were used for quantitative mean comparisons. A p-value of less than 0.05 was considered statistically significant.

RESULTS

Maternal and Neonatal Baseline Characteristics

A total of 105 neonates born to mothers with gestational diabetes mellitus were evaluated. The mean maternal gestational age was 37.54 \pm 1.80 weeks (range: 30.0 to 40.0 weeks). Term deliveries (>37 weeks) accounted for

81.0% (n = 85) of cases, while 19.0% (n = 20) were preterm (<37 weeks). The predominant mode of delivery was Lower Segment Caesarean Section (LSCS) in 48.6% (n = 51), followed by normal vaginal delivery in 43.8% (n = 46) and assisted delivery in 7.6% (n = 8). Late-onset GDM was diagnosed in 81.0% (n = 85) of mothers, while early-onset GDM was present in 19.0% (n = 20).

Among the 105 neonates, 50.5% (n = 53) were female and 49.5% (n = 52) were male. The mean birth weight was 2.97 \pm 0.67 kg (range: 1.40 to 4.50 kg). Weight classification for gestational age revealed that 53.3% (n = 56) were AGA, 33.3% (n = 35) were LGA, and 13.3% (n = 14) were SGA. The mean APGAR scores were 6.10 \pm 0.81 at 1 minute and 7.56 \pm 0.93 at 5 minutes.

Table 1: Maternal Baseline Demographics and Antenatal Variables (N = 105)

Variable	Category	Frequency (n)	Percentage (%)
Gestational Age	< 37 weeks (Preterm)	20	19.0%
	≥ 37 weeks (Term)	85	81.0%
Mode of Delivery	LSCS	51	48.6%
	Normal Vaginal Delivery	46	43.8%
	Assisted Delivery	8	7.6%
Onset / Duration of GDM	Early Onset	20	19.0%
	Late Onset	85	81.0%
Parity	Primigravida	44	41.9%
	Multigravida	61	58.1%
Antenatal Events	Uneventful	60	57.1%
	Previous LSCS	7	6.7%
	Cephalopelvic Disproportion	6	5.7%
	Antenatal Pericardial Effusion	1	1.0%

Table 2: Neonatal Baseline Characteristics (N = 105)

Variable	Category	Frequency (n)	Percentage (%)
Gender	Female	53	50.5%
	Male	52	49.5%
Birth Weight (kg)	< 1.99 kg	14	13.3%
	2.00 – 2.49 kg	6	5.7%
	2.50 – 2.99 kg	29	27.6%
	3.00 – 3.49 kg	25	23.8%
	3.50 – 3.99 kg	27	25.7%
	≥ 4.00 kg	4	3.8%
Weight for Gestation	Appropriate for GA (AGA)	56	53.3%
	Large for GA (LGA)	35	33.3%
	Small for GA (SGA)	14	13.3%
APGAR Score (1 min)	0 – 3	1	0.9%
	4 – 6	72	68.6%
	> 7	32	30.5%
APGAR Score (5 min)	4 – 6	16	15.2%
	> 7	89	84.7%

Spectrum of Cardiovascular Abnormalities

In the study cohort of 105 neonates born to GDM mothers, 2D Echocardiography confirmed normal cardiac anatomy and function in 90.5% (n = 95) of subjects. Cardiovascular abnormalities were documented in 9.5% (n = 10) of neonates.

The predominant cardiac lesion was Patent Ductus Arteriosus (PDA) associated with moderate-to-severe Pulmonary Arterial Hypertension (PAH), observed in 8 cases (7.6%). Asymmetric Septal Hypertrophy (ASH) / Hypertrophic Cardiomyopathy was diagnosed in 2 cases (1.9%). Coarctation of the Aorta was identified in 1 case (1.0%), and antenatal Pericardial Effusion was documented in 1 case (1.0%).

Table 3: Spectrum of 2D Echocardiographic Findings and Cardiovascular Abnormalities (N = 105)

Echocardiographic / Cardiovascular Finding	Frequency (n)	Percentage (%)
Normal 2D Echocardiogram	95	90.5%
Patent Ductus Arteriosus (PDA) with Moderate-to-Severe PAH	8	7.6%
Asymmetric Septal Hypertrophy (ASH)	2	1.9%
Coarctation of the Aorta	1	1.0%
Pericardial Effusion	1	1.0%
Total Cardiovascular Abnormalities	10	9.5%

Correlation of GDM Onset with Cardiovascular Morbidity and Mortality

Analysis of clinical outcomes demonstrated a direct relationship between the timing of maternal GDM onset and neonatal mortality. Neonates born to mothers with early-onset GDM experienced a significantly higher mortality rate (16.7%, n = 3) compared to those born to mothers with late-onset GDM (1.3%, n = 1) (Chi-square = 8.65, p = 0.003). The mean duration of NICU stay for infants requiring cardiovascular and intensive monitoring was 9.62 \pm 4.64 days. No statistically significant difference in the mean length of NICU stay was observed between early-onset (9.56 \pm 5.20 days) and late-onset GDM (9.63 \pm 4.54 days; p = 0.949).

Table 4: Association between GDM Onset and Neonatal Mortality Outcome (N = 97 Followed Cases)

Mortality Outcome	Early-Onset GDM (n = 18)	Late-Onset GDM (n = 79)	Total (N = 97)	p-value
Alive	15 (83.3%)	78 (98.7%)	93 (95.9%)	0.003*
Death	3 (16.7%)	1 (1.3%)	4 (4.1%)	

*Statistically significant (p < 0.05).

DISCUSSION

This prospective observational study provides critical clinical data on the spectrum of cardiovascular abnormalities in neonates born to gestational diabetic mothers admitted to a tertiary care hospital in Karnataka. In our study of 105 IDMs, 9.5% (n = 10) exhibited significant cardiovascular abnormalities, comprising PDA with moderate-to-severe PAH (7.6%), Asymmetric Septal Hypertrophy (1.9%), Coarctation of the Aorta (1.0%), and Pericardial Effusion (1.0%).

Comparing these findings with the study by Farah Zeba et al. (2025) conducted at KIMS Hospital, Bangalore, reveals important clinical parallels and phenotypic variations. Farah Zeba et al. reported a total cardiac defect rate of 83.9% among 56 echocardiographically evaluated IDMs, with Atrial Septal Defect (ASD) being the most prevalent anomaly (58.9%), followed by PDA (16.0%) and Ventricular Septal Defect (VSD, 3.6%). In contrast, our cohort demonstrated an overall cardiovascular abnormality rate of 9.5%, with Patent Ductus Arteriosus accompanied by moderate-to-severe Pulmonary Arterial Hypertension (7.6%) and Asymmetric Septal Hypertrophy (1.9%) forming the core spectrum.

Both studies highlight the central contribution of maternal diabetes to altered fetal and neonatal cardiovascular dynamics. While Farah Zeba et al. identified a higher frequency of simple interatrial shunts (ASD), our study underscores the clinical significance of ductal dependence, pulmonary vascular strain (PAH), and functional hypertrophic cardiomyopathy in infants of diabetic mothers requiring NICU admission.

Pathophysiological Mechanisms

The distinct spectrum of cardiac abnormalities observed in infants of diabetic mothers reflects dual pathophysiological mechanisms:

1. **Teratogenic Alterations in Organogenesis:** Early maternal hyperglycemia during the first trimester affects endocardial cushion tissue formation, heart looping, and neural crest cell migration, predisposing the fetus to structural defects like Coarctation of the Aorta, PDA, and septal anomalies.
2. **Hyperinsulinemic Myocardial Remodeling:** In mid-to-late gestation, persistent maternal glucose surges cross the placenta, inducing fetal hyperglycemia and pancreatic beta-cell hyperplasia. Fetal hyperinsulinemia activates myocardial insulin receptors, promoting hyperplastic

and hypertrophic responses within cardiac myocytes. This selective hypertrophy preferentially affects the interventricular septum, leading to Asymmetric Septal Hypertrophy (ASH). Severe septal hypertrophy can reduce left ventricular cavity size and cause dynamic left ventricular outflow tract obstruction.

Alignment with Global and Indian Literature

Our findings align with several regional and international clinical series:

- Shankar P et al. (2019) similarly identified Patent Ductus Arteriosus as the predominant cardiovascular anomaly in infants of diabetic mothers.
- Eisa RA & Babiker MS (2019) reported high rates of PDA (47.5%) and ASD (41.9%) among offspring of diabetic mothers evaluated by echocardiography.
- Gutgesell et al. (1976) and Breitweser et al. established that hyperinsulinemia drives cardiac septal hypertrophy and dynamic subaortic obstruction in IDMs, demonstrating that this hypertrophic cardiomyopathy typically regresses over the first few months of life as circulating insulin levels normalize.
- Rowland TW et al. documented a 4% incidence of structural CHD in IDMs, citing transposition of great arteries, VSD, and Coarctation of the Aorta as major findings.
- Chen L et al. (2019) confirmed in a systematic review that exposure to maternal diabetes significantly increases the overall risk of congenital cardiac defects in offspring.
- Hromadnikova I et al. (2020) demonstrated that children born to GDM pregnancies exhibit altered cardiovascular microRNA profiles linked to altered myocardial gene expression and structural heart defects.

Impact of GDM Duration and Onset

A major observation in our study was the statistically significant association between early-onset maternal GDM and adverse neonatal outcomes ($p = 0.003$). Mothers with early-onset GDM experience prolonged glycemic instability spanning early embryonic organogenesis and late fetal maturation. This extended exposure heightens the risk of both structural heart malformations and severe hemodynamic instability post-delivery.

Clinical Implications

The findings emphasize that physical examination alone is insufficient to exclude cardiovascular compromise in IDMs. Dynamic structural and functional lesions—such as asymmetric septal hypertrophy, pulmonary arterial hypertension, and coarctation of the aorta—may present subtly during the immediate newborn period. Routine screening via fetal echocardiography during the second trimester, followed by early postnatal 2D Echocardiography for all infants born to diabetic mothers, is vital for early identification, hemodynamic optimization, and reduction of adverse outcomes.

CONCLUSION

This study confirms that infants born to mothers with gestational diabetes mellitus face a heightened risk of cardiovascular abnormalities, predominantly Patent Ductus Arteriosus with Pulmonary Arterial Hypertension, Asymmetric Septal Hypertrophy, Coarctation of the Aorta, and Pericardial Effusion. Early onset of maternal GDM significantly amplifies the risk of severe cardiovascular compromise and neonatal mortality.

To minimize cardiovascular morbidity and mortality in infants of diabetic mothers, we recommend:

1. **Strict Antenatal Glycemic Control:** Optimizing maternal blood glucose levels prior to conception and throughout pregnancy.
2. **Universal Echocardiographic Screening:** Performing routine fetal echocardiography at 18–22 weeks of gestation and postnatal 2D echocardiography for all neonates born to diabetic mothers, regardless of clinical symptoms.
3. **Targeted Neonatal Management:** Ensuring prompt hemodynamic support and specialized NICU monitoring for infants identified with structural defects, pulmonary hypertension, or hypertrophic cardiomyopathy.

REFERENCES

1. Farah Zeba, Kumar MN, Chandrakala P, Shimpiger OS. Cardiac Anomalies in Infants of Diabetic Mothers: A Retrospective Observational Study in KIMS Hospital, Bangalore. Eur J Cardiovasc Med. 2025;15(7):684-689.
2. Reitzle L, Heidemann C, Baumert J,

Kaltheuner M, Adamczewski H, Icks A, Scheidt-Nave C. Pregnancy complications in women with pregestational and gestational diabetes mellitus. Dtsch Arztebl Int. 2023;120(6):81-88.

3. Loffredo CA. Epidemiology of cardiovascular malformations: prevalence and risk factors. Am J Med Genet. 2000;97(4):319-325.
4. Basu M, Garg V. Maternal hyperglycemia and fetal cardiac development: Clinical impact and underlying mechanisms. Birth Defects Res. 2018;110(20):1504-1516.
5. Negrato CA, Marques PR, Leite HB, Torigoe CN, Silva BF, Costa K, et al. Glycemic and nonglycemic mechanisms of congenital malformations in hyperglycemic pregnancies: a narrative review. Arch Endocrinol Metab. 2022;66(6):908-918.
6. Eisa RA, Babiker MS. Prevalence of VSD, PDA, and ASD in Saudi Arabia by echocardiography: a prospective study. J Diagn Med Sonogr. 2019;35(4):282-288.
7. Shankar P, Marol JS, Lysander SD, Manohar A. Cardiovascular malformations in infants of diabetic mothers: a retrospective study. Int J Contemp Pediatr. 2019;6(5):1998-2002.
8. Dawid G, Czeszynska MB, Horodnicka-Jozwa A, Wnuk W, Mojsiewicz M, Hnatyszyn G, et al. Secundum atrial septal defect in newborns of diabetic mothers. Med Wieku Rozwoj. 2006;10(3 Pt 1):355-364.
9. Dominic B, Mithun HK. Spectrum of congenital heart diseases in infants of diabetic mothers. Karnataka Paediatr J. 2025;40:19-22.
10. Sikarwar R, Hatkar PA. Study of fetal 2D echo in pregnant women with diabetes and gestational diabetes mellitus. Int J Reprod Contracept Obstet Gynecol. 2019;8(11):4214-4218.
11. Rowland TW, Hubbell JP, Nadas AS. Congenital heart disease in infants of diabetic mothers. J Pediatr. 1973;83(5):815-820.
12. Gutgesell HP, Speer ME, Rosenberg HS. Characterization of the cardiomyopathy in infants of diabetic mothers. Circulation. 1980;61(2):441-450.
13. Chen L, Yang T, Chen L, Wang L, Wang T, Zhao L, et al. Risk of congenital heart defects in offspring exposed to maternal diabetes mellitus: an updated systematic review and meta-analysis. Arch Gynecol Obstet. 2019;300(6):1491-1506.
14. Hromadnikova I, Kotlabova K, Dvorakova L, Krofta L, Sirc J. Substantially altered expression profile of diabetes/cardiovascular/cerebrovascular disease associated microRNAs in children descending from pregnancy complicated by gestational diabetes mellitus. Cells. 2020;9(6):1557.