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Original Article

Physiology Section

Echocardiographic Evaluation of Infants Born to Mothers with Gestational Diabetes Mellitus: A Cross-sectional Analytical Study

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ABSTRACT

Introduction: Gestational Diabetes Mellitus (GDM) is a chronic and progressive disease characterised by changes in the metabolism of carbohydrates, lipids, and proteins, leading to glucose intolerance. The foetal heart in GDM is affected throughout the gestational period, which may result in hyperplasia and hypertrophy of myocardial cells.

Aim: To determine the cardiac function and structural changes in infants of gestational diabetic mothers using Doppler-echocardiographic data.

Materials and Methods: A cross-sectional analytical study was conducted on 30 infants of mothers with GDM and 30 infants of non diabetic mothers at the Department of Physiology, R.G. Kar Medical College and Hospital, in collaboration with the Departments of Paediatric Medicine and Cardiology from January 2023 to August 2023. Neonatal screening echocardiography was performed by a cardiologist using a Philips Echocardiograph machine, Model: EPIQ 7C, Software Version: 4.0.2, with a linear convex probe of S8-3 MHz frequency to assess cardiac structure and function using 2D, M mode, and Conventional Doppler. The parameters studied included aortic root diameter (mm), left atrial diameter (mm), Interventricular Septal (IVS) in diastole (mm), left ventricular posterior wall thickness in diastole (mm), left ventricular internal diameter in diastole (mm), left ventricular internal diameter in end systole (mm), left ventricular fractional shortening (%), and left ventricular ejection fraction (%). Statistical analysis was performed using the Statistical Package for Social Sciences (SPSS) version-20.0 with an Independent Student's t-test.

Results: The mean gestational age for the cases was 38.50±2.57 weeks and 39.30±1.97 weeks for controls. The mean maternal age for cases was 25±4.37 years and for controls was 27±5.03 years. The left atrial diameter in cases (12.83±2.14 mm) was significantly smaller than that of the control (14.20±0.55 mm). The left ventricular posterior wall diameter in diastole was significantly increased in cases (4.53±0.50 mm) compared to controls (3.87±0.35 mm). Significant differences were observed in the interventricular septum in diastole {cases: 4.73±0.45 mm, control: 3.97±0.32 mm} and in the left ventricular internal diameter in end systole {cases: 12.30±1.84 mm, control: 13.83±1.02 mm} between cases and controls. Aortic root diameter was increased in 13.3% of cases. The percentage of left ventricular fractional shortening (cases: 35.47±3.74%, Control: 38.53±2.43%), Left Ventricular Ejection fraction (cases: 67.63±4.52%, control: 72.17±4.89%) were significantly decreased in cases compared to control.

Conclusion: The present study demonstrated the development of ventricular hypertrophy and compromised myocardial contractility in infants of diabetic mothers.

Keywords: Cardiac ultrasound, Infant of diabetic mothers, Ventricular hypertrophy

INTRODUCTION

GDM is defined as any degree of glucose intolerance that arises or is first detected during pregnancy. The prevalence of GDM is approximately 7.5% [1,2]. GDM is a chronic disease, and the underlying pathogenesis is similar to that observed in Type 2 Diabetes Mellitus (T2DM), involving a decrease in insulin sensitivity as gestation progresses [3]. It is characterised by changes in the metabolism of carbohydrates, lipids, and proteins, leading to glucose intolerance during pregnancy [4].

GDM impacts both the structure and function of the foetal heart, as well as foetal-placental circulation throughout the gestational period, due to the toxic effects of hyperglycaemia and hyperketonaemia. These effects modify multiple biochemical and signal transduction pathways, producing excessive free oxidative radicals that can impair autophagy, increase apoptosis, and disrupt cell homeostasis, proliferation, and migration of neural crest cells, which are crucial for the development of the heart and brain [5,6]. This may lead to diabetic fetopathy, altered placental villi vascularisation, and foetal venous thrombosis [7]. The proper expression of genes responsible for the correct development of the heart during embryogenesis is hindered in the early stages of pregnancy, potentially resulting in structural issues such as foetal cardiomyopathy, congenital cardiac malformations, and pathological foetal heart rates.

In the late gestational period, inadequate maternal glycaemic control and chronic intrauterine hyperglycaemia lead to foetal hyperinsulinaemia, increased total body weight, selective organomegaly, and myocardial hypertrophy [8-10]. Infants of diabetic mothers who use insulin during the third trimester of pregnancy are more likely to develop cardiovascular changes than newborns of non diabetic mothers, by a factor of 20.6 times [11]. Additionally, accelerated growth of the foetal heart occurs in the last two trimesters compared to foetuses of non diabetic mothers [12]. The presence of foetal and neonatal complications depends on various factors, including the type of diabetes, the level of HbA1c in early pregnancy, the degree and duration of hyperglycaemia and hyperketonaemia, maternal glycaemic control throughout pregnancy, and the presence of co-morbid conditions [13].

Doppler echocardiography or cardiac ultrasound is a non invasive tool used to assess cardiac function. This tool allows for the determination of cardiac function and structural changes in Infants of gestational Diabetic Mothers (IDM) [14]. Due to the scarcity of studies in Eastern India, this study was conducted with an aim to

determine the cardiac function and structural changes in infants of gestational diabetic mothers during the postnatal period through Doppler echocardiographic data.

MATERIALS AND METHODS

A cross-sectional analytical study was conducted among 30 infants of diagnosed GDM mothers and 30 infants of non diabetic mothers in the Department of Physiology in collaboration with the Department of Paediatric Medicine and Department of Cardiology at R.G. Kar Medical College and Hospital, Kolkata, West Bengal, India from January 2023 to August 2023. Neonatal screening echocardiography was performed by a Cardiologist. The study was conducted after obtaining Institutional Ethical Clearance from the R.G. Kar Medical College Ethics Committee (IEC no- RKC/612).

Inclusion criteria:

- For Cases: a) Infants of diabetic mothers; b) Either gender; c) Ages between zero months to four months; d) Term and preterm babies
- For Controls: a) Infants of non diabetic mothers; b) Either gender; c) Ages between zero months to four months old, those who came for immunisation and regular follow-up in the well-baby clinic in the Outpatient Department (OPD).

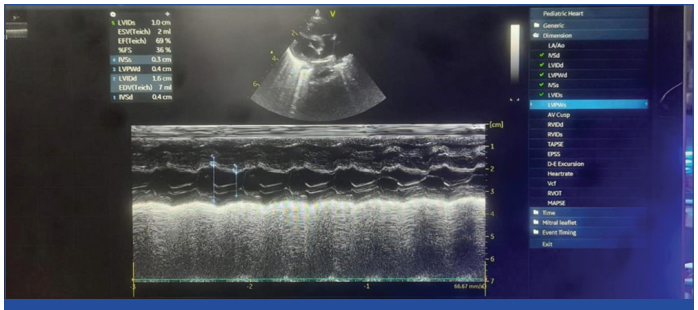
Exclusion criteria:

- For Infants: Chromosomal abnormalities, evidence of congenital foetal anomalies (including cardiac), foetal arrhythmia, intrauterine growth restriction, active illness in infants, and restless babies.
- For Maternal: Infants of mothers with pre-existing hypertension, gestational hypertension, haematologic diseases, liver disease, renal disease, maternal cardiac diseases.

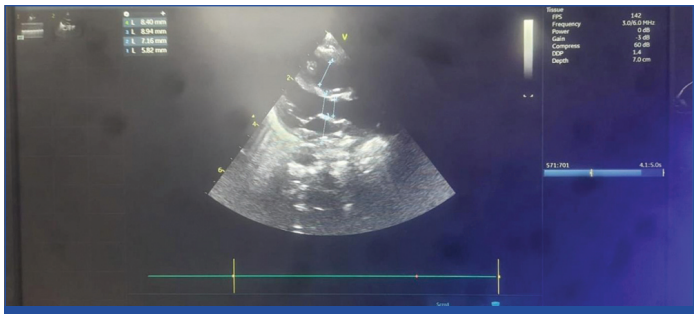
Study Procedure

The Philips Echocardiograph machine, Model: EPIQ 7C, Software Version: 4.0.2, with a linear convex probe of S8-3 MHz frequency to assess cardiac structure and function using 2D, M mode, and conventional Doppler was utilised for the study. Screening echocardiography was conducted after obtaining written consent from individual mothers following the inclusion and exclusion criteria. A comprehensive history of both the mother and infant, complete clinical examination, and anthropometric measurements were performed before the procedure, and the cases and controls were matched for age, sex, and weight. The infants were calm and quiet, and no anaesthesia was used.

The parameters studied included: Aortic root diameter (mm), left atrial diameter (mm), Interventricular Septal thickness (IVS) in Diastole (mm), left ventricular posterior wall thickness in diastole (mm), left ventricular internal diameter in diastole (mm), left ventricular internal diameter in end systole (mm), left ventricular fractional shortening (%), left ventricular ejection fraction (%), Left Ventricular mass (LV mass) (g), Left Ventricular Mass Index (LVMI) (g/m^2), Relative Wall Thickness (RWT) {2x posterior wall thickness (mm)/left ventricular diameter in end systole(mm)}. [Table/Fig-1] displays the left ventricular internal diameter, ejection fraction, fractional shortening, interventricular septal thickness, left ventricular posterior wall thickness, left ventricular internal diameter in end systole and end diastole and [Table/Fig-2] shows the aortic root diameter in 2-D and M-mode echocardiography.



[Table/Fig-1]: Cardiac parameters in an infant of diabetic mother in a 2-D and M-mode echocardiography.



[Table/Fig-2]: Aortic root diameter in an infant of diabetic mother in a 2-D and M-mode echocardiography.

Assessment of left ventricular function was performed by calculating the percentage of ejection fraction [15] and the percentage shortening of the internal dimension [16]. The percentage shortening of the internal dimension equals the Left Ventricular internal dimension at the End of Diastole (LVEDD) minus that at the end of systole (LVESD) divided by the dimension of the left ventricle at the end of diastole (LVEDD) and multiplied by 100 {(LVEDD-LVESD/LVEDD)×100}. The calculation of ejection fraction was done by applying the same formula except that all values (except 100) are cubed {(LVEDD^3- LVESD^3/LVEDD^3)×100}.

The left ventricular mass was calculated by cubing the sum of the thickness of the interventricular septum, the left ventricular internal dimension, and the thickness of the left posterior wall (all at the end of diastole), subtracting the cubed value for LVEDD, and multiplying by 1.05 [17]. Septal hypertrophy was defined as a septal thickness of 5 mm or more [18]. LVMI was measured by calculating {LV mass/Body Surface Area (BSA)} (Software Omni Calculator).

STATISTICAL ANALYSIS

Statistical analysis was conducted using SPSS version 20.0, employing an Independent Student's t-test.

RESULTS

Age, Period Of Gestation (POG), Corrected Gestational Age (CGA), birth weight and length of the baby were matched in both groups [Table/Fig-3]. In the present study, 14 (46.7%) of the babies were boys, and 16 (53.3%) were girls [Table/Fig-4].

Variables	Case (n=30) (Mean±SD)	Control (n=30) (Mean±SD)	p-value
Age (days)	59.63±22.82	58.67±26.27	0.88
Period Of Gestation (POG) (weeks)	38.50±2.57	39.30±1.97	0.18
Corrected Gestational Age (CGA) (weeks)	44.23±24.69	51.20±23.81	0.27
Birth weight (kg)	2.83±0.59	2.87±0.35	0.79
Length of the baby (cm)	56.43±3.65	54.53±3.75	0.052

[Table/Fig-3]: Baseline characteristics of the study subjects. Independent student t-test was used

Variables		Case (n=30)	Control (n=30)
Gender	Boy	14 (46.7)	13 (43.3)
	Girl	16 (53.3)	17 (56.7)
Term/ Preterm	Term	22 (73.3)	26 (86.7)
	Preterm	8 (26.7)	4 (13.3)
Mother's treatment history	Diet	17 (56.7)	
	Insulin	7 (23.3)	
	Metformin	5 (16.7)	
	Both insulin and metformin	1 (3.3)	

[Table/Fig-4]: Distribution of subjects according to gender, term and preterm and mother's treatment history.

There was a significant difference in the left atrial diameter, interventricular septum in diastole, left ventricular posterior wall diastole, and left ventricular internal diameter end systole. Left ventricular contractility was assessed by measuring left ventricular fractional shortening and left ventricular ejection fraction, where both parameters were significantly decreased among the cases compared to the control. There was a significant increase in RWT, left ventricular mass, and LVMI among the cases compared to the control [Table/Fig-5].

Variable	Case (n=30) (Mean±SD)	Control (n=30) (Mean±SD)	p-value
Aortic root diameter (mm)	7.60±0.97	7.93±0.25	0.73
Left atrial diameter (mm)	12.83±2.14	14.20±0.55	0.01*
Interventricular septum in diastole (mm)	4.73±0.45	3.97±0.32	0.01*
Left ventricular posterior wall thickness in diastole (mm)	4.53±0.50	3.87±0.35	0.01*
Left ventricular internal diameter in diastole (mm)	18.77±2.71	19.67±0.88	0.09
Left ventricular internal diameter in end systole (mm)	12.30±1.84	13.83±1.02	0.01*
Left ventricular fractional shortening (%)	35.47±3.74	38.53±2.43	0.01
Left ventricular ejection fraction (%)	67.63±4.52	72.17±4.89	0.01*
Relative Wall Thickness (RWT)	0.5±0.09	0.38±0.02	<0.0001*
Left ventricular mass (g)	13.73±3.07	11.32±2.03	0.0007*
Left Ventricular Mass Index (LVMI) (g/m^2)	58.42±14.19	46.47±6.90	0.0001

[Table/Fig-5]: Comparison of echocardiography findings between cases and control.
Independent Student's t-test was used

The present study found that 30 (100) of the cases had an intact interventricular septum, 28 (93.3%) of the cases had an intact interatrial septum, but 2 (6.7%) of the cases had an interatrial septal defect. Patent ductus arteriosus was absent in 30 (100) of the cases. The left ventricular internal diameter end diastole increased in 2 (6.7) and decreased in 5 (16.6%) of the cases, and a normal left ventricular internal diameter end systole was seen in 29 (96.7%) of the cases [Table/Fig-6].

Variables		Case (n=30)	Control (n=30)
Interventricular septum (mm)	Intact	30 (100)	30 (100)
	Defect	0	0
Interatrial septum (mm)	Intact	28 (93.3)	29 (96.7)
	Defect	2 (6.7)	1 (3.3)
Patent ductus arteriosus (mm)	Present	0	0
	Absent	30 (100)	30 (100)
Aortic root diameter (mm)	Normal	26 (86.7)	30 (100)
	Increased	4 (13.3)	0
Left atrial diameter (mm)	Normal	27 (90)	30 (100)
	Increased	3 (10)	0
Left ventricular posterior wall diameter in diastole (mm)	Normal	10 (33.3)	30 (100)
	Increased	20 (66.7)	0
Left ventricular internal diameter in end diastole (mm)	Normal	23 (76.7)	30 (100)
	Increased	2 (6.7)	0
	Decreased	5 (16.6)	0
Left ventricular internal diameter in end systole (mm)	Normal	29 (96.7)	30 (100)
	Increased	1 (3.3)	0

[Table/Fig-6]: Distribution of echocardiography findings among the cases and control.

DISCUSSION

The present study demonstrates a significant increase in left ventricular posterior wall thickness in diastole among infants of gestational diabetes mothers compared to non diabetic mothers (p-value <0.01). However, Deorari AK et al., showed that there is increased thickness of the left ventricular posterior wall in systole in IDM (infants of diabetic mothers) compared to controls [19]. The study by Garg S et al., supports the findings of the present study [20]. A study by El-Gansoury MM et al., revealed that nine out of 69 IDM had both left ventricular posterior wall hypertrophy and interventricular septal hypertrophy with suboptimally glycaemic control [21]. In the present study, it was observed that 20 (66.7%) of cases had an increased left ventricular posterior wall thickness.

In the present study, there was a significant (p-value <0.01) increase in the interventricular septum among the cases compared to the control. The study by Deorari AK et al., study was consistent with the present study [19]. Conversely, another study by Fouda UM et al., suggests that the interventricular septum was significantly thicker in pre-existing diabetic mothers compared to controls [22]. A research by El-Gansoury MM et al., which supports the current study, found that 21 out of 69 IDM babies had interventricularseptal hypertrophy [21]. Intact interventricular septum was observed in 30 (100%) of cases in the present study. In this study, an interatrial septal defect was present in 2 (6.7%) of cases. A study by Abu-Sulaiman RM and Subaih B found that 5% of cases had an interatrial septal defect (ASD) [23]. A study by El-Gansoury MM et al., discovered that among 69 infants of diabetic mothers, three (4.3%) neonates had congenital heart disease, two of whom had an atrial septal defect [21].

A study by Deorari AK et al., showed that hypertrophy of the septum occurred in three out of seven babies with hypoglycaemia, but in only five out of 24 without hypoglycaemia, and in none of the control babies, suggesting an association between neonatal hypoglycaemia and septal thickness [19]. Another study by Breitweser JA et al., also demonstrated this association [24], where they found that five out of 18 IDM had disproportionate interventricular septal hypertrophy, and profound hypoglycaemia (serum glucose concentration <20 mg/dL) was present among those five infants.

During the late gestational period, inadequate maternal glycaemic control results in chronic intrauterine hyperglycaemia, which may lead to foetal hyperinsulinaemia. As a consequence of foetal hyperinsulinaemia, there may be an increase in total body weight and selective organomegaly. For example, the heart may be affected as it is an insulin-sensitive organ, and there may be an increased expression as well as affinity of insulin receptors in the cardiac cells [8]. Along with the degree of maternal and foetal hyperglycaemia and concomitant foetal hyperinsulinaemia, the interventricular septal pathology is also related to the glycogen deposition of the septum [25].

Left ventricular contractility was assessed by measuring left ventricular fractional shortening (%) and left ventricular ejection fraction (%). Both parameters were significantly decreased in cases compared to the control in the present study. In contrast, the study by Deorari AK et al., found higher left ventricular ejection fraction and left ventricular fractional shortening in infants of diabetic mothers compared to the control [19]. Kosak Barany A et al., suggest slightly higher fractional shortening in the cases than in the control (p-value=0.04) [26]. A study by El-Gansoury MM et al., detected impaired left ventricular contractility (FS <36%) in 75.4% of infants of diabetic mothers [21].

Present study found a significant decrease in the left atrial diameter among cases compared to the control group (p-value <0.01). A study by Deorari AK et al., suggests that there was no significant difference in left atrial diameter measurements between cases and controls [19]. The left ventricular internal diameter in end systole was significantly decreased in cases compared to the control group (p-value <0.01) in the present study, whereas Deorari AK et al., did not find any significant differences in the left atrial diameter between cases and controls [19]. A study by El-Gansoury MM et al., on the contrary, found that the left ventricular diameter in end systole was within normal limits [21]. In the present study, the aortic root diameter did not showed any significant difference between cases and controls, and the aortic root diameter was increased among 4 (13.3%) of cases. A study by Walther FJ et al., found that the mean aortic root diameter was smaller among infants of diabetic mothers than in the control group [27], which contradicts the findings of the present study. Similarly, the left ventricular internal diameter in diastole measurement was taken, which did not show any significant difference between cases and controls in the present study. However, the left ventricular internal diameter in diastole was increased among 2 (6.7%) of cases, whereas it was decreased among 5 (16.6%) of cases. On the contrary, a study by El-Gansoury MM et al., found that the left ventricular diameter at end diastole was within the normal range [21].

Present study revealed that the left ventricular mass (LV mass), LVMI (LV mass/BSA), and RWT were significantly increased among the cases compared to the control group. Kosak Barany A et al., described increased LV mass and LV mass/BSA ratio in infants of gestational diabetic mothers (p-value <0.05) [26], and Deorari AK et al., found significantly increased left ventricular mass in infants of mothers with gestational diabetes [19]. These findings are likely due to the large number of insulin receptors in this cardiac area [19,22]. According to Gardiner HM in the foetus of a diabetic mother, hypertrophic cardiomyopathy may not be a primary cardiac dysfunction but a functional adaptive process [28]. Sielinsky P et al., suggest that transient myocardial hypertrophy may be seen in infants of diabetic mothers, which may disappear within about six months to two years after birth [29]. A study by Reller MD et al., suggests that control and early treatment of diabetes mellitus in pregnancy improve the clinical outcome, but the development of the disease cannot be prevented [30].

It was an analytical study in which the majority of the confounding factors were excluded. It provides meaningful information to predict the structural or functional cardiac complications of the offspring of gestational diabetic mothers. All IDMs should undergo early screening echocardiography to prevent any cardiac complications. Maternal glycaemic control and early intervention may limit the consequences of IDMs. In the future, further research with a larger sample size and follow-up study is recommended. The present study will help in planning how to alleviate the consequences of GDM on the offspring.

Limitation(s)

This study was conducted with a small sample size and limited study area, and a follow-up study could not be performed.

CONCLUSION(S)

The present study showed that infants of mothers with GDM develop ventricular hypertrophy and compromised myocardial contractility due to cardiac remodeling. Further follow-up is required to assess whether the cardiac changes found in the study are regressive or deteriorating in nature. Therefore, all infants born to diabetic mothers should undergo screening echocardiography to prevent any complications.

REFERENCES

[1] Lawrence JM, Contreras R, Chen W, Sacks DA. Trends in the prevalence of preexisting diabetes and gestational diabetes mellitus among a racially/ethnically diverse population of pregnant women, 1999-2005. Diabetes Care. 2008;31(5):899-904.

[2] Albrecht SS, Kuklina EV, Bansil P, Jamieson DJ, Whiteman MK, Kourtis AP, et al. Diabetes trends among delivery hospitalisations in the U.S., 1994-2004. Diabetes Care. 2010;33(4):768-73.

[3] Creasy and Resnik's. Maternal-Fetal Medicine: Principles and Practice- 7th Editon. Available from: https://www.elsevier.com/books/creasy-and-resniks-maternal-fetal-medicine-principlesand-pracce/resnik/978-1-4557-1137-6.

[4] American Diabetes Association. Gestational diabetes mellitus report of the expert committee on the diagnosis and classification of diabetes mellitus. Diabetes Care. 2004;27(suppl_1):S88-90.

[5] Kumar SD, Dheen ST, Tay SS. Maternal diabetes induces congenital heart defects in mice by altering the expression of genes involved in cardiovascular development. Cardiovasc Diabetol. 2007;6:34.

[6] Helle E, Priest JR. Maternal obesity and diabetes mellitus as risk factors for congenital heart disease in the offspring. J Am Heart Assoc. 2020;9:e011541.

[7] Corrigan N, Brasil DP, McAuliffe F. Fetal cardiac effects of maternal hyperglycaemia during pregnancy. Birth Defects Res A Clin Mol Teratol. 2009;85(6):523-30.

[8] Boucher J, Kleinridders A, Kahn CR. Insulin receptor signaling in normal and insulinresistant states. Cold Spring Harb Perspect Biol. 2014;6(1):a009191.

[9] Elmekkawi SF, Mansour GM, Elsafty MS, Hassanin AS, Laban M, Elsayed HM. Prediction of fetal hypertrophic cardiomyopathy in diabetic pregnancies compared with postnatal outcome. Clin Med Insights: Womens Health. 2015;8:39-43.

[10] Wang X, Lian Y, Wang X, Tian M. Study of regional left ventricular longitudinal function in fetuses with gestational diabetes mellitus by velocity vector imaging. Echocardiography. 2016;33(8):1228-33.

[11] Costa HD, mãe diabética Recém nascido de, Procianoy RS, Leone CR, editors. Programa de atualisac¸ão em neonatologia (PRORN)---Sociedade Brasileira de Pediatria. 2008. p.09-43. Porto Alegre.

[12] Schwarts R, Gruppuso PA, Petsold K, Brambilla D, Hiilesmaa V, Teramo KA. Hyperinsulinemia and macrosomia in the fetus of the diabetic mother. Diabetes Care. 1994;17(7):640-48.

[13] Paine LL, Greener DL. Obstetrics: Normal and problem pregnancies. Edited by: Steven G. Gabbe, Jennifer R. Niebyl, and Joe L. Simpson. New York: Churchill Livingstone, 1986. Journal of Nurse-Midwifery. 1988:33(6):290-91.

[14] Bogo MA, Pabis JS, Bonchoski AB, Santos DCd, Pinto TJ, Simões MA, et al. Cardiomyopathy and cardiac function in fetuses and newborns of diabetic mothers. Jornal de Pediatria. 2021;97(5):520-24.

[15] Feigenbaum H. Echocardiography. 2nd ed. Philadelphia: Lea and Febiger, 1976.

[16] Way GL, Wolfe RR, Eshaghpour E, Bender RL, Jaffe RB, Ruttenberg HD. The natural history of hypertrophic cardiomyopathy in infants of diabetic mothers. J Pediatr. 1979;95(6):1020-25.

[17] Bennet DH, Evans DW. Correlation of left ventricular mass determined by echocardiography with vector cardiographic and electrocardiographic voltage measurements. Br Heart J. 1974;36(10):981-87.

[18] Avery Gordon B. Neonatology: Pathophysiology and management of the newborn. 3rd ed. Philadelphia: JB Lippincott, 1987:342-3.

[19] Deorari AK, Saxena A, Singh M, Shrivastava S. Echocardiographic assessment of infants born to diabetic mothers Archives of Disease in Childhood. 1989;64:721-24.

[20] Garg S, Sharma P, Sharma D, Behera V, Durairaj M, Dhall A. Use of fetal echocardiography for characterisation of fetal cardiac structure in women with normal pregnancies and gestational diabetes mellitus. J Ultrasound Med. 2014;33(8):1365-69.

[21] El-Gansoury MM, El-Masry SA, El-Farrash RA, Anwar M, Abd Ellatife RS. Infants of diabetic mothers: Echocardiographic measurements and cord blood IGF-I and IGFBP-1. Pediatric Diabetes. 2012;13(2):189-96.

[22] Fouda UM, Abou EL, Kassem MM, Hefny SM, Fouda RM, Hashem AT. Role of fetal echocardiography in the evaluation of structure and function of fetal heart in diabetic pregnancies. J Matern Fetal Neonatal Med. 2013;26(6):571-75.

[23] Abu-Sulaiman RM, Subaih B. Congenital heart disease in infants of diabetic mothers: Echocardiographic study. Pediatric Cardiology. 2004;25(2):137-40.

[24] Breitweser JA, Meyer RA, Speriling MA, Tsang RC, Kaplan S. Cardiac septal hypertrophy in hyperinsulinemic infants. J Pediatr.1980;96(3 Pt 2):535-39.

[25] Nold JL, Georgieff MK. Infants of diabetic mothers. Pediat Clin N Ame. 2004;51(3):619-37.

[26] Kosak Barany A, Jokinen E, Kero P, Touminen J, Ronnemaa T, Valimaki I. Impaired left ventricular diastolic function in newborn infants of mothers with pregestational or gestational diabetes with good glycaemic control. Early Hum Dev. 2004;77(1-2):13-22.

[27] Walther FJ, Siassi B, King J, Wu PY. Cardiac output in infants of insulin-dependent diabetic mothers. J Pediatr. 1985;107(1):109-14.

[28] Gardiner HM. Response of the fetal heart to changes in load: From hyperplasia to heart failure. Heart. 2005;91(7):871-73.

[29] Sielinsky P, da Costa MH, Oliveira LT, Bonow FP, da Silva NI, Hagemann LL. Natural history of myocardial hypertrophy and its association with hyperinsulinism in infants of diabetic mothers. Arq Bras Cardiol. 1997;69(6):389-94.

[30] Reller MD, Tsang RC, Meyer RA, Braun CP. Relationship of prospective diabetes control in pregnancy to neonatal cardiorespiratory function. J Pediatr. 1985;106(1):86-90.

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