The Relationship between Serum Glycated Albumin in Diabetic Pregnant Women and Hypertrophic Cardiomyopathy in Their Neonates

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ABSTRACT

Background: Maternal diabetes mellitus elevates the risk for a range of maternal-fetal complications.
Objective: This study aimed to assess the correlation between serum glycated albumin (GA) concentrations in diabetic pregnant females and the development of hypertrophic cardiomyopathy (HCM) in their neonates, and to evaluate the validity of GA as an early predictor for HCM. **Patients and methods:** This cross-sectional research was performed on 44 diabetic pregnant women and their neonates at Suez Canal University Hospital. Maternal glycated albumin was measured during the third trimester. Neonatal echocardiography was performed within 14 days postpartum to assess cardiac function, ventricular size, and wall thickness. **Results:** Of the 44 neonates, 15 (34.1%) had hypertrophic cardiomyopathy. Mothers of neonates with HCM had significantly longer diabetes duration (14.1 ± 6.2 against 9.2 ± 3.4 years, p=0.013) and greater mean GA levels (24.5 ± 2.9 against 15.3 ± 1.99, p<0.001) compared to mothers of neonates without HCM. A significant direct correlation was found between GA and interventricular septum thickness in systole (r=0.649, p<0.001). ROC curve analysis illustrated that GA at a cut-off value of ≥ 20.1 had sensitivity of 92.9% and specificity of 96.2% for predicting HCM in neonates of diabetic mothers.
Conclusions: Serum glycated albumin level is a clinically effective glycemic control marker with high predictive value for hypertrophic cardiomyopathy in infants of diabetic mothers. GA monitoring may serve as a valuable tool for diabetes management during pregnancy to reduce the risk of cardiac complications in neonates.
Keywords: Gestational diabetes, Glycated albumin, Hypertrophic cardiomyopathy, Infant of diabetic mother, Pregnancy.

INTRODUCTION

In neonates, hypertrophic cardiomyopathy constitutes a notable co-morbidity, manifesting as myocardial hypertrophy of one or both ventricular walls, which frequently includes the interventricular septum. This condition is further accompanied by significant impairment of both systolic and diastolic function. Its prevalence is estimated at approximately 40% in babies born to mothers with diabetes, with 5% of these cases presenting with clinical symptoms [(1)].

Maternal diabetes mellitus elevates the risk for a range of maternal-fetal complications. During the initial trimester, maternal hyperglycemia may induce diabetic embryopathy, leading to significant congenital anomalies and spontaneous abortions [(2)]. In the subsequent 2nd and 3rd trimesters, persistent maternal hyperglycemia precipitates a cascade of fetal metabolic disturbances. This includes fetal hyperglycemia, which in turn stimulates excessive insulin secretion from the fetal pancreas, a state defined as fetal hyperinsulinemia [(3)]. The transplacental glucose gradient can result in fetal blood glucose concentrations reaching as high as 234 mg/dl. This state of fetal hyperinsulinemia is a primary etiological factor for numerous neonatal morbidities including macrosomia, hypocalcemia, polycythemia, hyperbilirubinemia, delayed pulmonary maturation, and myocardial hypertrophy specifically the development of HCM in infants of diabetic mothers is strongly hypothesized to be a direct consequence of fetal hyperinsulinemia acting on the myocardium [(1)].

In the monitoring of glycemic status, Glycated hemoglobin (HbA1c) is established as the principal biomarker, providing a retrospective measure of mean blood glucose concentrations over the preceding one to two months [(4)]. For glycemic management during pregnancy, the Scottish Intercollegiate Guideline Network (SIGN) advocates for the maintenance of HbA1c levels within a near-normal glycemic range (equivalent to 81.0–126 mg/dl) [(5)].

A key advantage of GA over HbA1c in the context of pregnancy is its independence from hemoglobin metabolism. HbA1c concentrations can be artifactually altered by physiological changes in late pregnancy, such as iron deficiency anemia, whereas GA levels remain uncorrelated with hemoglobin concentrations [(6)]. This distinction has led to the proposal that GA may eventually supersede HbA1c as the standard for evaluating glycemic control [(7)].

This research has been performed to evaluate the association among maternal diabetes and hypertrophic cardiomyopathy in infants utilizing GA as a marker for glycemic control to enhance the neonatal results of diabetic mothers and to validate the utility of GA as an indicator of glycemic control throughout gestation.

PATIENTS AND METHODS

A cross-sectional research was performed on 44 cases of diabetic pregnant females and their neonates at the Gynecology and Obstetrics Department and Neonatal Intensive Care Unit (NICU) at Suez Canal University Hospital in Ismailia.

Inclusion criteria: Full-term neonates of diabetic mothers. **Exclusion criteria:** Pregnant women taking medications or having conditions that affect albumin metabolism like nephrotic syndrome, hyperthyroidism, obesity, and steroid use. Neonates with multiple

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congenital anomalies or cardiac anomalies, neonates with congenital heart disease and neonates with hypertension.

Mothers were subjected to: Full history, physical examination and glycated Albumin (GA) was measured approximately two weeks before labor. For GA, ELISA kits were used and programmed onto a Siemens Advia1800 Automated Chemistry Analyzer based on the manufacturer's instructions [8]**.** GA reference range (Normal Range: 11-16) [9].

Neonates were subjected to full history, physical examination and echocardiography was performed using a focused ECHO of Philips type to assess cardiac functions, ventricular size, and wall thickness for neonates within 14 days post-partum.

Echo parameters included: Interventricular septal end diastole (IVSd)**,** LV end-diastolic diameter (LVEDD)**,** Left ventricular posterior wall thickness in diastole (LVPWd)**,** Interventricular septal end systole (IVSs)**,** LV end-systolic diameter (LVESD)**,** Ejection fraction (EF)**,** Fractional shortening (FS)**,** Septum to Post wall ratio (Sep/Post W)**,** Left ventricular outflow tract obstruction (LVOTO), Left atrium to left ventricle outflow tract diameter (LA/LVOTD)**,** Left ventricle outflow tract gradient (LVOT grad)**,** Left ventricle outflow tract velocity time integral (LVOT VTI)**,** Stroke volume (SV)**,** Heart rate (HR)**,** Cardiac output (CO) and Cardiac index (CI).

Additional assessments included: LVSV = LVOTD × LVOT VTI**,** CO = SV × HR and CI = CO × BSA.

Ethical Approval: The research was permitted through the Ethics Committee of Faculty of Medicine, Suez Canal University (approval code: SUEC/2023/158). All participants gave written informed consent before enrolment. The study adhered to the Helsinki Declaration throughout its execution.

RESULTS

The mean age of the mothers was 35.32 ± 3.01 years, and the mean body mass index was 31.13 ± 5.14 kg/m². The mean period of diabetes mellitus was 14.1 ± 6.2 years, with 70.5% of women receiving insulin therapy and 56.8% having diabetes-related complications (Tables 1).

Table (1): Maternal features of the research population

Variables	**Number=44**	
Maternal age (years)	35.32 ± 3.01	
median(range)	35(30-44)	
Weight (kg)	77.3 ± 10.47	
median(range)	74(55-99)	
Height (m)	1.54 ± 0.19	
median(range)	1.5(1.4-1.8)	
BMI (kg/m²)	31.13 ± 5.14	
median(range)	32(19-41)	
Duration of DM (years)		14.1 ± 6.2
Type of treatment	O.H	13 (29.5%)
	Insulin	31 (70.5%)
Complications	25 (56.8%)	

The study included 19 (43.2%) male and 25 (56.8%) female neonates. The mean birth weight was 3.19 ± 0.60 kg, mean length was 49.92 ± 2.88 cm, and mean body surface area was 0.21 ± 0.02 m². Cesarean section was the mode of delivery for 59.1% of neonates (Table 2).

Table (2): Neonatal features of the research population

Variable		**Number=44**
Gender	Male	19 (43.2%)
	Female	25 (56.8%)
Weight (kg)		3.19 ± 0.60
Length (cm)		49.92 ± 2.88
BSA (m²)		0.21 ± 0.02
Mode of delivery	Normal vaginal	18 (40.9%)
	CS	26 (59.1%)
BSA=body surface area, CS=Cesarean section		

Echocardiographic parameters of the study population demonstrated that among the 44 neonates, 15 (34.1%) had hypertrophic cardiomyopathy (HCM), with 10 (22.7%) had non-obstructive HCM and 5 (11.4%) had hypertrophic obstructive cardiomyopathy (HOCM) (Table 3).

Table (3): Echo parameters of IDM in the study population

Parameter	**N=44**
IVSd (mm)	4.17 ± 0.21
LVEDD (mm)	10.93 ± 1.06
LVPWd (mm)	3.71 ± 0.33
IVSs (mm)	4.07 ± 0.38
LVESD (mm)	0.32 ± 0.11
LVPWs (mm)	0.33 ± 0.25
Sep/Post W (mm)	1.92 ± 0.34
LVOTD (mm)	11.11 ± 1.07
LA/LVOTD (mm)	0.97 ± 0.21
LVOT grade (mm)	3.80 ± 1.02
LVOT VTI (mm)	9.87 ± 1.21
SV	0.43 ± 0.06
CO	0.49 ± 0.09
CI	2.67 ± 0.35
FS	34.7 ± 2.7
EF	55.22 ± 8.88
IVSd: interventricular septum in diastole, LVPWd: left ventricle posterior wall in diastole, IVSs: interventricular septum in systole, LVPWs: left ventricle posterior wall in systole, Sep/Post W: Septum to Post wall ratio,	
SV= LVEDD × LVSd, CI = CO / BSA, SV: Stroke volume, CI: Cardiac Index, LVEDD: left ventricle end diastolic diameter, LVSd: interventricular septum in diastole, CO: cardiac output, BSA: body surface area, FS=Fractional Shortening	

There were no statistically significant variances in maternal weight, age, height, and BMI between mothers of neonates with and without HCM. However, mothers of neonates with HCM had significantly longer duration of diabetes (14.1 ± 6.2 against 9.2 ± 3.4 years, p-value equal to 0.013) compared to mothers of neonates without HCM (Table 4).

Table (4): Comparison of maternal characteristics of the study groups

Variables	**Neonates with Hypertrophi Cardiomyopathy (N=15)**		**Neonates withou Hypertrophic Cardiomyopathy (N=29)**	**P-value**
Maternal age (years)	35.5 ± 2.9		35.7 ± 2.9	0.894
Weight (kg)	78.1 ± 8.8		73.1 ± 11.6	0.171
Height (m)	1.6 ± 0.20		1.5 ± 0.19	0.130
BMI (kg/m²)	30.1 ± 5.5		32.1 ± 4.3	0.075
Duration of DM		14.1 ± 6.2	9.2 ± 3.4	0.013*
Type of treatment	O.H	5 (33.3%)	8 (27.6%)	0.082
	Insulin	10 (66.7%)	21 (72.4%)	

*Statistically significant as p-value below 0.05.

A statistically significant distinction was observed between neonates with and without HCM in terms of interventricular septum thickness in systole (IVSs), which was higher in neonates with HCM (5.03 ± 0.15 vs. 4.56 ± 0.29, p-value below 0.001). Insignificant variances were observed in other echo parameters. Neonates with HCM had significantly higher mean glycated albumin levels (24.5 ± 2.9) compared to neonates without HCM (15.3 ± 1.99, p-value below 0.001) (Table 5).

Table (5): Comparative analysis of Echo parameters and glycated hemoglobin in neonates with and without hypertrophic cardiomyopathy

Parameter	**Neonates with Hypertrophic Cardiomyopathy (N=15)**	**Neonates without Hypertrophic Cardiomyopathy (N=29)**	**P-value**
IVSd (mm)	4.02 ± 0.15	3.99 ± 0.26	0.724
LVPWd (mm)	3.13 ± 0.31	3.26 ± 0.36	0.248
IVSs (mm)	5.03 ± 0.15	4.56 ± 0.29	<0.001*
LVPWs (mm)	0.38 ± 0.26	0.31 ± 0.25	0.417
Sep/Post W (mm)	1.39 ± 0.34	1.36 ± 0.46	0.860
LVEED (mm)	10.7 ± 1.07	11.4 ± 1.04	0.471
LVESD (mm)	0.31 ± 0.12	0.33 ± 0.11	0.395
LVOTD (mm)	11.33 ± 1.17	10.99 ± 0.84	0.346
LA/LVOTD (mm)	1.04 ± 0.02	0.93 ± 0.21	0.107
LVOT grade (mm)	3.89 ± 0.98	3.75 ± 1.06	0.683
LVOT VTI (mm)	9.78 ± 1.08	9.92 ± 1.28	0.744
GA (%)	24.5 ± 2.9	15.3 ± 1.99	<0.001*

A statistically significant direct moderate correlation was observed among glycated albumin and interventricular septum thickness in systole (IVSs) (r=0.649, p-value below 0.001). ROC curve analysis demonstrated that GA at a cut-off value of ≥ 20.1 had a specificity of 96.2% and sensitivity of 92.9% for predicting hypertrophic cardiomyopathy in neonates of diabetic mothers, with an area under the curve (AUC) of 0.912 (figure 1).

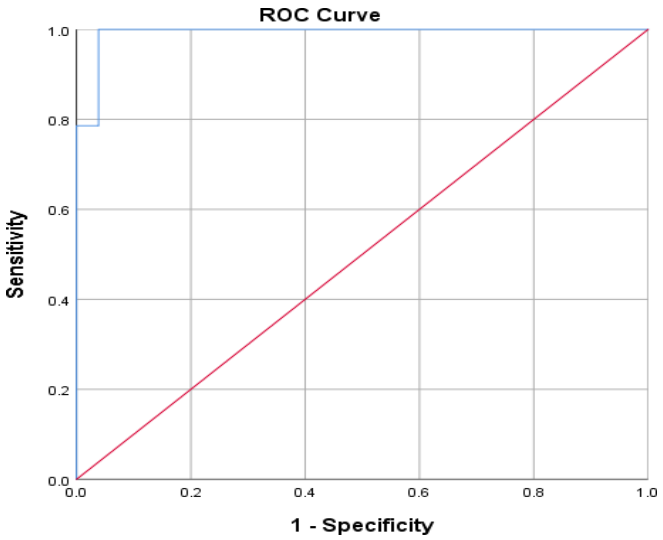

Figure (1): ROC curve of GA in prediction of hypertrophic cardiomyopathy among the study neonates.

DISCUSSION

As the foremost endocrine disorder complicating gestation, maternal diabetes mellitus is increasingly prevalent, paralleling the rising incidence of type 2 diabetes, obesity, and metabolic syndrome in the female reproductive-aged population [(10)]. Notwithstanding significant progress in antenatal monitoring and clinical management, unfavorable perinatal and enduring health consequences for both the maternal and offspring dyad persist as substantial clinical challenges. A prominent complication within this context is hypertrophic cardiomyopathy (HCM) found in infants of diabetic mothers (IDMs). This cardiac pathology is defined by an asymmetrical hypertrophy that predominantly involves the interventricular septum and/or the ventricular free walls. Characteristically, HCM in this specific infant population presents as a transient phenomenon, with a spontaneous regression of cardiac hypertrophy and normalization of myocardial dimensions typically occurring within the initial months of postnatal life [(11)].

One significant neonatal complication is hypertrophic cardiomyopathy (HCM), a condition where the heart muscle thickens. Unlike the progressive and poor-prognosis HCM in infants of non-diabetic mothers, HCM in IDMs typically resolves within a few months after birth [(12, 13)]. Fetal hyperinsulinism, resulting from maternal hyperglycemia, is thought to cause this cardiac issue by stimulating the growth of myocardial cells [(14)]. The mothers in the study had a long history of diabetes, poor glycemic control (mean HbA1c of 8.1 ± 0.98%), and a high rate of complications. The study found that 34.1% of the neonates developed HCM, a figure consistent with previous research which reported rates from 30% to 43.5% [(15, 16)].

Infants of diabetic mothers face a greater possibility of cardiovascular problems due to prenatal hyperglycemia and other factors [(17, 18)]. Poor glycemic control is linked to altered cardiac function [(19)]. Even newborns who appear asymptomatic may have uninvestigated cardiac issues [(20)]. The study's findings are supported by a research showing that IDMs with HCM have lower fractional shortening (FS), a key measure of left ventricular function [(21)] and that even asymptomatic IDMs can have altered LV function [(22)]. There is some debate whether these cardiac changes are linked to the baby's weight or are a direct result of the metabolic environment in the womb [(23, 24)].

Poor maternal glycemic control is strongly associated with HCM.

The study revealed that mothers of infants with HCM had significantly higher HbA1c levels. This aligns with other studies that have consistently linked high maternal HbA1c with the development of HCM in their infants [(15, 16, 24, 25)]. Conversely, good glycemic control is associated with normal fetal heart growth [(26)], although some research has found increased cardiac size even with careful metabolic management [(27)].

The study highlighted glycated albumin (GA) as a valuable marker for predicting neonatal cardiac outcomes. Neonates with HCM had significantly higher maternal GA levels (24.5 ± 2.0) compared to normal neonates (15.3 ± 1.99). GA is considered a useful indicator during pregnancy because it reflects shorter-term glucose levels (two to three weeks), is more closely related to post-meal glucose levels, and isn't influenced through alterations in hemoglobin metabolism, which can make HbA1c less reliable in late pregnancy [(28-30)]. This is supported by another study that also found significantly greater GA in mothers of infants with myocardial hypertrophy [(31)]. Japanese studies have confirmed GA as a better marker for blood glucose control [(32)] and recommend its use, noting that a GA value ≥15.8% is related to a greater frequency of neonatal complications [(33, 34)].

The study determined that a maternal GA cut-off value of ≥ 20.1 had a 96.2% specificity and 92.9% sensitivity for predicting HCM in neonates of diabetic mothers. This finding is similar to research that identified a comparable predictive cut-off value [(31)].

CONCLUSION

GA is an excellent predictor for monitoring gestational diabetes and maternal glycemic control. It can predict hypertrophic cardiomyopathy in neonates of diabetic mothers who are at great possibility during pregnancy.

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