Annals of Neonatology Journal

OPEN ACCESS
ISSN: 2636-3596

Original Article

Cord Blood Hemoglobin A1c and MCP-1 as Predictors for Cardiomyopathy in Infants of Mothers with Gestational Diabetes

Abdel-Azeem M. El-Mazary[1]; Madiha Sayed[1]; Hazem Ali[2]; Waleed Abdel-Hameed[3]; Nancy Tawadros[*1]

DOI: 10.21608/ANJ.2019.45815

***Correspondence:** Pediatric Department, Faculty of Medicine, Minia university, Egypt
Email: nancytawadros@yahoo.com
Full list of author information is available at the end of the article.

Abstract

Background: Diabetes mellitus is one of the most common metabolic disorders that disturb the health of mothers during pregnancy and their babies. MCP-1 is a chemokine accountable for the calling of inflammatory cells. It was linked to diminished cardiac function. HbA1c level is also considered a marker of follow-up and treatment in diabetes mellitus. **Objectives**: The study intended to measure the levels of cord blood Hemoglobin A1c and MCP-1and correlate their levels with the cardiomyopathic changes and other echocardiographic parameters in infants of diabetic mothers **Patients and methods**: it is a descriptive cross-sectional comparative study which comprised 110 term neonates separated into two groups, the first group included 70 neonates born to diabetic mothers (IDMs) and 40 healthy neonates born to non-diabetic mothers as a control group. This study was conducted from April 2016 to April 2017 at Minia University Hospital for Obstetric and children. During the first week of life, all babies were evaluated for medical history, thorough clinical examination with a focus on cardiac examination, and laboratory investigations which included CBC and neonatal blood sugar as routine investigations& HbA1c and MCP-1 as specific investigations in addition to echocardiographic assessment. **Results:** IDMs have increased cardiac dimensions, impaired diastolic function, and lower fractional shortening compared to the control group. Cord MCP-1and HbA1c were significantly high in IDMs and their levels significantly correlated with the echocardiographic parameters in IDMs. The sensitivity and specificity of HbA1c in detecting cardiomyopathy was 83.3% and 95% with positive and negative predictive value 83.7% and 88.1% awhile for MCP-1 sensitivity and specificity were 93.3% and 97.5%, positive and negative predictive values were93.1 %and 95.1%. **Conclusion:** MCP-1 and HbA1c were found to be excellent predictors for cardiomyopathy in infants of diabetic mothers with gestational diabetes.

Key words: MCP-1, HbA-1c, IDMs, cardiomyopathy

Introduction

The prevalence of cardiomyopathy (HCM) particularly interventricular septal hypertrophy (IVSH) ranges from 10% to 71% [1]. Upregulation of Monocyte chemoattractant protein-1 (MCP-1) occurs under different inflammatory conditions and considered the main signal for the monocytes/macrophages accumulation in various diseases. In patients with dilated cardiomyopathy (DCM), MCP-1 was detected in endomyocardial biopsy samples and the degree of cardiac function impairment was linked to MCP-1 expression levels in the myocardium [2].

Glycated hemoglobin (HbA1c), which reflects glycemic state over the previous 1-2 months .It is used as an indicator for recent control of blood sugar and treatment marker in diabetic patients [3]. European Association for the Study of Diabetes, the International Diabetes Federation, and the American Diabetes Association defined levels of HbA1c at 7% or more is associated with increased risks of cardiovascular diseases, diabetic nephropathy, neuropathy and retinopathy [4]. The chronic exposure of the fetus to maternal hyperglycemia is closely linked to fetal macrosomia and various cardiac anomalies and cardiomyopathy changes [1].

Methods

The present study was a descriptive cross-sectional comparative one that included 110 full-term neonates divided into two groups, the first group included70 full-term neonates born to diabetic mothers, and the second group included 40 full-term apparently healthy neonates born to non-diabetic mothers as a control group. This study was conducted from April 2016 to April 2017 at Minia University Hospital for Obstetric and children.

Term infants borne to diabetic mothers were eligible to be included in the study except those with evidence of sepsis based on clinical or laboratory findings, babies with suspected or confirmed

inborn error of metabolism and those with major congenital malformation other than cardiac one. For enrolled neonates, complete history taking included the maternal illness with a focus on diabetes Mellitus specifically gestational type, maternal medication, parity, mode of delivery, gestational age and APGAR score. Detailed clinical examination was done and included birth weight, respiratory, cardiac, abdominal and neurological examination.

A routine investigation like complete blood count and random blood sugar were done in addition to assay of hemoglobin A1c (HbA1c) and monocyte chemotactic protein-1(MCP-1). MCP-1 was measured in the hospital laboratory by enzyme-linked immunoassay (ELISA) method that allows for in vitro quantitative determination of Human Monocyte Chemotactic Protein 1 (MCP-1). The concentration of MCP-1 in the samples was determined by comparing the O.D of the samples to the standard curve. Assessment of HbA1c: HbA1c is boronate affinity in this method aminophenyl boronic acid reacts specifically with the cis-diol groups of glucose bound to Hb.

Echocardiography Examination was performed by a pediatric cardiologist having experience in echocardiography according to the recommendations of The American Society of Echocardiography [5] and by the use of SonoSite, M-turbo system with P10x 8-MHZ transducer (multifrequency transducer) (SonoSite Fujifilm Inc., Bothell Wa, USA). The examination consisted of M-mode, 2-D, pulsed, continuous-wave, and color Doppler blood flow velocity measurements of the heart valves. Parameters like aortic valve diameter(AOV),fractional shortening(FS) , ejection fraction (EF) ,interventricular septal end-diastolic thickness (IVSd), interventricular septal end-systolic thickness(IVSs), left ventricular end-diastolic dimension (LVDd), left ventricular end-systolic dimension (LVDs), left ventricular posterior wall

thickness (LVPW) and right ventricular end-diastolic dimension (RVDd) were taken.

Ethical considerations

The study was revised and approved by the scientific committee of the pediatrics department, Minia University. Written and verbal consent was obtained from the parents of babies prior to inclusion in the study.

Statistical analysis

Analysis of data was done using Statistical Program for Social Science version 20 (SPSS Inc., Chicago, IL, USA). Quantitative variables were described in the form of mean ±standard deviation and range. Qualitative variables were described as a percent. In order to compare quantitative variables between two groups, Correlation studies were done using Spearman's rank correlation coefficient. P-value <0.05 was considered significant in all analyses.

Results

Table (1) showed that infants of diabetic mothers (IDMs) had a significantly higher birth weight than the control group with p value< 0.001, and while delivery by C.S in IDMs was significantly higher when compared to the control group with p value< 0.04. As regarding gestational age and sex distribution, there was an insignificant difference between the two groups.

Table (2) showed that there was a significant increase in levels of cord blood HbA1c, MCP-1, hemoglobin and hematocrit in IDMs (P<0.024, 0.027, 0.041, and 0.001 respectively) when compared with controls also there was significant hypoglycemia in IDMs in comparison to the control group (P<0.05), while there was insignificant difference in neonatal white blood cells and neonatal platelets count between the two studied groups.

Table (3) showed that an Increase in IVSd, IVSs, LVPW, LVDd, LVDs and RVDd measures of IDMs when compared with the control group, also there was a decrease in EF% and FS % of IDMs when compared with the control

group however insignificant difference was found in AVO (aortic valve diameter) between the two groups.

Table (4) should that HbA1c correlated significantly with LVPW r=, 0.298. (p=0.012.). Another high significant correlation between HbAIc and each of IVSd, IVSs, LVDs, LVDd, RVDd and AOV were noticed where (r = 0.724, 0.544, 0.544 0.433and0.673) respectively (p-value <0.000). A significant negative correlation was noticed between HbA1c and ES and EF where r = (-0.551- & -0.688 respectively) (p<0.000).MCP-1 was strongly associated with each of IVSd. IVSs, LVPW, LVDs and RVDd and AOV where (r= o.793, 0.687, 0.465, 0.557, o.548 and 0.437) (p<0.000). Also, negative highly significant correlations were noticed between MCP-1 and each of FS AND EF where r=-0.663 and -0.664 p < 0.000).

The sensitivity and specificity of HbA1c in the prediction of cardiomyopathy at cutoff point > 4.7% were 83.3%, 95%. AUC was 0.93 while that of MCP-1 in detecting cardiomyopathy in IDM babies was Sensitivity 93.3%, specificity was 97.5% at cutoff point >390 pg/ml, AUC was 0.96 (figure 1&2).

Discussion

Fetuses and newborns of mothers with GDM are vulnerable to Cardiac complications in the form of congenital heart malformation and ventricular hypertrophy which represent the major causes of morbidity and mortality [6]. The incidence of hypertrophic cardiomyopathy (HCM), particularly interventricular septal hypertrophy (IVSH), ranges from 10%to 71% [1]. Regarding the demographic data, birth weight was significantly high in IDMs in comparison to controls in the present study. These results can be explained by the fact that maternal diabetes and/or obesity affects this pathway through exaggeration of the physiological insulin resistance which develops during pregnancy, which in turn contributes to maternal high blood sugar and dyslipidemia that leads to increased

nutrient delivery to the fetus, subsequently resulting in fetal hyperinsulinemia and macrosomia [6].

In our study cesarean section was the predominant mode of delivery in IDMs, this was inconsistent with Rafiq W et al, *2015* who reported similar results [7]. Similarly, Mathew et al described an increased incidence of cesarean section in macrosomic neonates [8]. Hemoglobin and hematocrit values showed a significant increase in diseased babies when compared to controls(table2). These results were near to that done by Cetin et al., *2011* [9] who found that hemoglobin, HbF levels, and venous neonatal hematocrit values in IDMs were significantly high when compared to controls. Metzger et al, *2010* reported that IDMs have higher levels of RBCs and consequently hemoglobin and hematocrit levels; they explained these findings by the positive effect of fetal insulin on erythropoiesis [10].

There was significant hypoglycemia in IDMs when compared with the control group, this was in agreement with Mimouni et al, *2013* who showed that the prevalence of hypoglycemic episodes in IDMs is as high as 40% when compared with the control group [11]. This metabolic risk is supposed to be a result of relative fetal hyperinsulinism, manifested as a feedback mechanism that opposed the high glucose levels induced by maternal diabetes [12]. In our study, we found that there were insignificant differences in WBCs count and platelet count between IDMs and control groups, which was in agreement with Pilgaard et al, *2010* who reported that there was insignificant difference in WBCs and platelets numbers between IDM and control group [13], however another study reported that WBCs and platelets values were high in IDMs which can be explained by chronic acidosis and hypercapnia in diabetic pregnancies in addition to different patients criteria and sample size [14]. Cord blood HbA1c was significantly higher in IDMs than healthy normal neonates. These results were in

agreement with several previous reports ,Davison et al showed that glycated hemoglobin levels were significantly higher in IDMs compared to infants of non-diabetic mothers [15], similarly Koja et al, *2011* reported that the levels of glycated hemoglobin collected from infant borne to diabetic mothers were higher than that of healthy neonates [16].

In the present study, there was a significant rise in levels of cord blood MCP-1 in IDMs when compared to healthy controls, and this was similar to the study of Vuguin et al, *2013*who mentioned that MCP-1 levels in IDMs were high when compared to control and which can be a result of hyperglycemic state reflected by higher HbA1c levels in diabetic patient and the presence of the acetoacetate ketone which may be a factor in the increased MCP-1 levels in IDMs cord blood [17]. Another study found that MCP-1 is elevated during pregnancy and also augmented in GDM compared to non-GDM, this increased MCP-1 levels may lead to adverse pregnancy outcomes [18]. In contrast with our study, Kurepa et al,*2012* showed that MCP-1 levels in cord- blood of mothers with gestational diabetes was similar to healthy mothers, this may be related to small sample size as they studied all types of diabetes (gestational, type 1 diabetes, and type 2 diabetes) [19].

In the present study, there was a significant correlation between birth weight and cord blood HbA1c. This matches with a previous study which reported that elevated HbA1c was a reliable predictor for poor pregnancy outcomes, especially macrosomia; they attributed this to hyperinsulinemia which has a strong anabolic effect [20].

Our study showed that there was a significant positive relationship between birth weight and cord blood MCP-1, and this was in agreement with Lappas et al, *2011* who reported MCP-1 is a pro-inflammatory cytokine that is expected to play a key role in the development of insulin resistance, and so may play a part in the fetal overgrowth and maybe

enhanced fat deposition seen in infants whose mothers have GDM [20].

However, Kurepa et al, *2012* reported that there was no relationship between blood MCP-1 levels and fetal birth weight, thus, differences in gestational age or fetal birth weight are unlikely to have any effect on changes in MCP-1[19].

As regarding echocardiographic measures in this study, there was a significant increase in cardiac dimensions (except aortic valve diameter) in IDMs than the control group, and impaired systolic and diastolic function in IDMs than control group in the form of a lower value of FS% and this matches with El-Ganzoury et al,*2012* who reported a highly significant progressive increase in IVSd, IVSs, LVPW, LVDd, LVDs, and RVDd with the increase in birth weight, whereas, aortic valve diameter (AOV) and ejection fraction showed insignificant differences between the three studied groups [21]. Similarly, Korraa et al,*2012* found a significant abnormality in the left atrial thickness and inter-ventricular septal dimension in IDMs in comparison to control [22].

In contrast with our study, Katheria et al,*2012* reported that the thickness of the interventricular septum between the controls and IDMs were similar[(23], also Demirorem et al,*2005* reported no differences in the echocardiographic measurements of macrosomic and non-macrosomic IDMs, they suggested that cardiac changes are not only due to presence of macrosomia or glucose-insulin metabolism but rather to the combination of macrosomia, glucose-insulin metabolism, genetic and maternal anthropometric factors [24].

Our findings revealed that HbA1c has high sensitivity and specificity for detecting cardiomyopathy and there was a highly significant correlation between cord blood HbA1c and all echocardiographic measurements. We noticed that cord blood HbA1c in IDMs was inversely correlated with EF percent

and FS percent. El-Ganzoury*2012* et al observed that 26 of the 30 newborns with HCM had a Hb-A1c of less than 8% (8–9.1), while the remaining four had a HbA1c of less than 6.5 percent [21]. Ullmo et al, 2007 also identified the association between poor maternal glycemic management and hypertrophic cardiomyopathy [25]. In contrast with our study Pradhan et al, *2007* reported that HbA1c remained a strong predictor of diabetes but without association with cardiovascular diseases, these results can be attributed to the difference in gestational ages and weights of patients in their study [26].

Our findings showed that MCP-1 has high sensitivity and specificity for detecting cardiomyopathy and there was a highly significant correlation between cord blood MCP-1 and all echocardiographic measurements. We also discovered that cord blood MCP-1 in IDMs was inversely correlated with EF percent and FS percent, which agrees with the findings of Kobayashi et al, *2008* where the serum levels of MCP-1 in patients with dilated cardiomyopathy were significantly elevated compared with those of healthy control subjects and the expression level was inversely correlated with left ventricular ejection fraction (LVEF) [27]. Also, Iwasaki et al reported that in HCM patients' levels of MCP-1 are negatively correlated with left ventricular fractional shortening which was determined by echocardiography [28].

Limitations for this study: were the following up of those IDMs with regard to their echocardiographic measures as well as the serial measurement of neonatal glucose and maternal HbA1c during the third trimester.

Conclusions

HbA1c and MCP-1 were good predictors for occurrence of cardiomyopathy in Infants of diabetic mothers with gestational diabetes.

Acknowledgements

To all NICU staff members Minia University Hospital for Obstetrics and children

Author's contributions

SS and EA conceived the study. ME revised the patients' medical reports and the final manuscript. All authors revised the final draft of the manuscript

Conflict of interest

The authors have no conflict of interests to declare.

Funding

This study received no special funding and was totally funded by the authors.

Author's details

[1]Pediatric Department, Faculty of Medicine, Minia University, Egypt

[2]Cardiology Department, Faculty of Medicine, Minia University, Egypt.

[3]Clinical-Pathology Department, Faculty of Medicine, Minia University, Egypt

Date received: 13th March, 2019, accepted 28th April, 2021

References

1. Stuart A, Amer-Wåhlin I, Gudmundsson S, Marsál K, Thuring A, Källen K. Ductus venosus blood flow velocity waveform in diabetic pregnancies. Ultrasound Obstet Gynecol 2010; 36(03): 344–349.
2. Dusi V, Ghidoni A, Ravera A, De Ferrari GM, Calvillo L. Chemokines and Heart Disease: A Network Connecting Cardiovascular Biology to Immune and Autonomic Nervous Systems. Mediators Inflamm 2016; 59029472.
3. Gillett MJ. International Expert Committee report on the role of the A1c assay in the diagnosis of diabetes: Diabetes Care 2009; 32(7):1327-1334.
4. Seino Y, Nanjo K, Tajima N, Kadowaki T, Kashiwagi A, Araki E, Ito C, Inagaki N, Iwamoto Y, Kasuga M. Committee of the Japan Diabetes Society on the Diagnostic Criteria of Diabetes Mellitus: Report of the committee on the classification and diagnostic criteria of diabetes mellitus. J Diabetes Investig 2010; 1:212-228.
5. Skinner J. Normal Doppler ultrasound measurements in the newborn. In: Skinner J, Alverson D, Hunter S, eds. 435. Echocardiography for the Neonatologist. Edinburgh: Churchill Livingstone: 2000; 73–86.
6. O'brien CM, Poprzeczny A, Dodd JM. Implications of maternal obesity on fetal growth and the role of ultrasound. Exp Rev Endocrinol Metab. (2017) 12:45–58. 10.1080/17446651.2017.1271707.
7. Wasim Rafiq, Sheik Quyoom Hussain, Muzzafer Jan, Bilal Ahmad Najar.

Clinical and metabolic profile of neonates of diabetic mothers. International Journal of Contemporary Pediatrics; 2015; (2):114-8.

8. Mathew M, Machado L, Al-Ghabshi R, Al-Haddabi R. Fetal macrosomia. Risk factor and outcome. Saudi Med J. 2005; 26(1):96-100.
9. Cetin H, Yalaz M, Akisu M, Kultursay N. Polycythaemia in Infants of Diabetic Mothers: β-Hydroxybutyrate Stimulates Erythropoietic Activity. J Int Med Res. 2011; 39:815–21.
10. Metzger BE, Persson B, Lowe LP, Dyer AR, Cruickshank JK, Deerochanawong C, Halliday HL, Hennis AJ, Liley H, Ng PC, Coustan DR, Hadden DR, Hod M, Oats JJ, Trimble ER. Hyperglycemia and adverse pregnancy outcome study: neonatal glycemia. Pediatrics 2010; 126: e1545-e1552.
11. Mimouni FB, Mimouni G, Bental YA. Neonatal management of the infant of diabetic mother. Pediatrics &Therapeutics 2013; 4:186.
12. Nold JL, Georgieff MK. Infants of diabetic mothers. Pediatric Clinics of North America. 2004; 51,619-637.
13. Pilgaard K, Faerch K, Carstensen B, Poulsen P, Pisinger C, Pedersen O, Witte DR, Hansen T, Jorgensen T, Vaag A. Low birth weight and premature birth are both associated with type 2 diabetes in a random sample of middle-aged Danes. Diabetologia 2010; 53: 2526–2530.
14. RAJ R. Prediction of neonatal hypoglycemia using cord blood C-peptide and cord HbA1c in Infants of Diabetic Mother; The Antiseptic 2016; 113, (1): 28 - 30.
15. Davison AS, Green BN, Roberts NB. Diabetes in pregnancy: effect on glycation and acetylation of the different chains of fetal and maternal hemoglobin. Clinical Biochemistry 2011; 44:198–202.
16. Koga M, Murai J, Saito H, Yamada Y , Mori T, Suno S, Takeuchi K, Suzuki S, Fujieda K , and Kasayama S. Measurement of glycated hemoglobin and glycated albumin in umbilical cord: evaluation of the glycemic control indicators in neonates. J Perinatol 2011; 31: 430–433.
17. Vuguin PM, Hartil K, Kruse M, Kaur H, Lin CLV, Fiallo A , Alan Scott Glenn, Avanee Patel, Lyda Williams, Yoshinori Seki, Ellen B. Katz, Maureen J. Charron. Shared effects of genetic and intrauterine

and perinatal environment on the development of metabolic syndrome. PLoS One 2013; 8:e63021.

18. Klein K, Satler M, Elhenicky M, Brix J, Krzyzanowska K, Schernthaner G. Circulating levels of MCP-1 are increased in women with gestational diabetes. Prenat Diagn 2008; 28:845–51.
19. Kurepa, D., Pramanik, A.K., Kakkilaya, V., Caldito, G., Groome, L.J., Bocchini, J.A. and Jain, S.K. Elevated acetoacetate and monocyte chemotactic protein-1 levels in cord blood of infants of diabetic mothers. Neonatology 2012; 102: 163-168. 5.
20. Lappas M, Hiden U, Desoye G, Froehlich J, Hauguel-de Mouzon S, Jawerbaum A. The role of oxidative stress in the pathophysiology of gestational diabetes mellitus. Antioxid Redox Signal. 2011; 15:3061–100.
21. El Ganzoury M.M., EL Masry S.A., EL Farrash R.A., Anwar M. and Ellatife R.Z.. Infants of diabetic mothers; echocardiographic measurements and cord blood IGF-1 and IGF binding protein-1. Pediatr. Diabetes 2012; 13: 189-196
22. Korraa A, Ezzat MH, Bastawy M, Aly H, El-Mazary AA, Abd El-Aziz L. Cardiac troponin I levels and its relation to echocardiographic 2012.
23. Katheria A, Leone T. Altered transitional circulation in infants of diabetic mothers with strict antenatal obstetric management: a functional echocardiography study. J Perinatol. 2012; 32(7):508–513
24. Demiroren K, Cam L, Oran B, et al. Echocardiographic measurements in infants of diabetic mothers and macrosomic infants of non diabetic mothers. J Perinat Med 2005; 33(03) :232–235.
25. Ullmo S, Vial Y, Di Bernardo S, Roth-Kleiner M, Mivelaz Y, Sekarski N, Ruiz J, Meijboom EJ. Pathologic ventricular hypertrophy in the offspring of diabetic mothers: a retrospective study. Eur Heart J 2007; 28: 1319-1325.
26. Pradhan AD, Rifai N, Buring JE, Ridker PM. Hemoglobin A1c predicts diabetes but not cardiovascular disease in nondiabetic women. Am J Med 2007; 120:720–727.
27. Kobayashi M, Nakamura K, Kusano KF, Nakamura Y, Ohta-Ogo K, Nagase S, et al. Expression of monocyte chemoattractantprotein-1 in idiopathic dilated cardiomyopathy. Int J Cardiol

2008; 126:427–9.

28. Iwasaki J., Nakamura K., Matsubara H., Nakamura Y., Nishii N., Banba K., Murakami M., Ohta-Ogo K., Kimura H., Toh N., Nagase S., Oka T., Morita H., Kusano K. F., Ohe T. Cardiovasc. Pathol. 2009; 18, 317-322.

Table 1: Neonatal demographic data for Infants of diabetic mothers (IDMs) and control groups.

Item	**Group I IDMs (n=70)**	**Group II Control (n=40)**	**P value**
Gestational age(Weeks) Mean ± SD Range	38.88 ± 0.843 (40 - 38)	38.1 ± 0.901 (40 – 38)	0.186
Birth weight (Kg) Mean ± SD Range	3.45 ± 0.535 (2.6 - 4.2)	2.9 4 ± 0.228 (2.35 - 3.4)	0.001**
Mode of delivery NVD CS	13 (19 %) 57 (81 %)	15 (38 %) 25 (62%)	0.041*
Sex Male Female	32 (45.7 %) 38 (54.3 %)	20 (50 %) 20 (50 %)	0.527

Cs, caesarean section; NVD, spontaneous vaginal delivery; SD, standard deviation.
*: significant difference at p value < 0.05

Table 2 : Studied laboratory data of IDMs and control groups.

Item	Group I IDMs (n=70)	Group II Control (n=40)	P value
Neonatal HB (gm %) Mean ± SD Range	15.5 ± 0.96 (14 – 17)	13.9 ± 0.65 (12 – 15)	0.041*
Neonatal Hematocrit (%) Mean ± SD Range	47.9 ± 2.68 (40.5 – 57)	43.7 ± 2.20 (35 – 46)	0.001**
Neonatal WBCs (cu.mm× 10^9 / L) Mean ± SD Range	11 ± 3.26 (5.5 – 13)	10.7± 2.98 (5 – 12.4)	0.082
Neonatal Platelets (1000's /cu.mm) Mean ± SD Range	270 ± 1.43 (225 – 310)	268 ± 1.63 (216 – 304)	0.164
Neonatal blood glucose (mg/dl) Mean ± SD	34.6±11.3	77.2±19.8	0.05*
Cord blood HbA1c (%) Mean ± SD Range	7.49 ± 0.95 (5.7 – 8.9)	4.218±0.165 (4.0 – 4.4)	0.027*
Cord blood MCP-1 Mean ± SD Range	389.4 ± 97.5 (243 – 590)	200.85 ± 37.94 (104 – 280)	0.024*

Student' test to compare between mean of two groups of numerical (Parametric) data.
Chi square test for qualitative data between groups
*: significant difference at p value < 0.05
HB, hemoglobin ; WBCs, White blood cells; HbA1c, glycated hemoglobin A1c; Monocyte chemotactic protein-1 (MCP-1).

Table 3: echocardiographic data in IDMs and controls

Item	Group I IDMs (n=70) Mean ± SD	Group II Control (n=40) Mean ± SD	P value
IVSd(mm)	4.157 ± 0.63	3.140 ± 0.11	0.007**
IVSs(mm)	4.906 ± 0.54	4.22 ± 0.91	0.001**
LVPW(mm)	3.85 ± 1.27	3.20 ± 0.37	0.002**
LVDd(mm)	16.2 ± 1.3	15.1± 0.97	0.0035**
LVDs(mm)	11.4 ± 1.04	9.120 ± 0.53	0.001**
RVDd(mm)	41.62± 2.47	37.3 ± 2.24	0.007**
AOV(mm)	9.54 ± 1.15	9.13 ± 0.854	0.418
FS (%)	36.11 ± 5.31	41.2 ± 0.992	0.041*
EF (%)	69.12 ±3.98	73.53 ± 4.71	0.049*

AOV, aortic valve diameter; FS, fractional shortening ; EF, ejection fraction; IVSd, interventricular septal end-diastolic thickness; IVSs, interventricular septal end-systolic thickness; LVDd, left ventricular end-diastolic dimension; LVDs, left ventricular end-systolic dimension; LVPW, left ventricular posterior wall thickness; RVDd, right ventricular end-diastolic dimension; SD, standard deviation.

Chi square test for qualitative data between groups

*: significant difference at p value < 0.05

Table 4: Correlation between cord blood HbA1c, cord blood MCP – 1, and echocardiographic measures and birth weight in IDMs group .

Item	Cord blood HbA1c		Cord blood MCP - 1	
	r	p	r	p
IVSd	0.724	0.000*	0.793	0.000*
IVSs	0.605	0.000*	0.687	0.000*
LVPW	0.298	0.012*	0.465	0.000*
LVDd	0.433	0.000*	0.252	0.035*
LVDs	0.544	0.000*	0.557	0.000*
RVDd	0.604	0.000*	0.548	0.000*
AOV	0.673	0.000*	0.000*	0.000*
FS (%)	-0.551-	0.000*	-0.666-	0.000*
EF (%)	-0.688-	0.000*	-0.666-	0.000*
Birth weight	0.706	0.000	0.734	0.000

*: significant difference at p value < 0.05

.

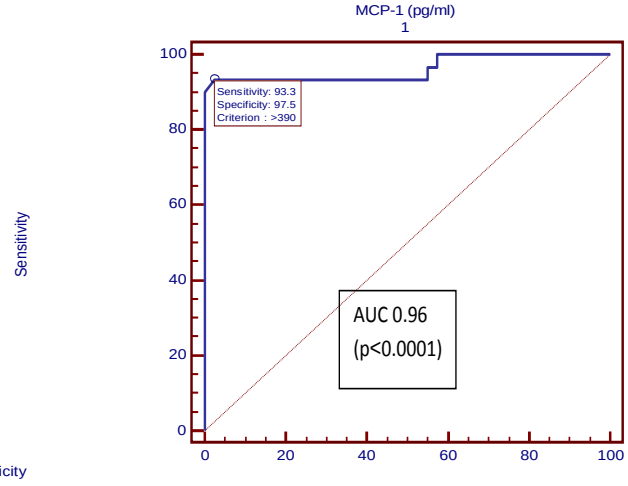

Figure 1: Receiver operating characteristic (ROC) curve of the MCP-1 for prediction of cardiomyopathy

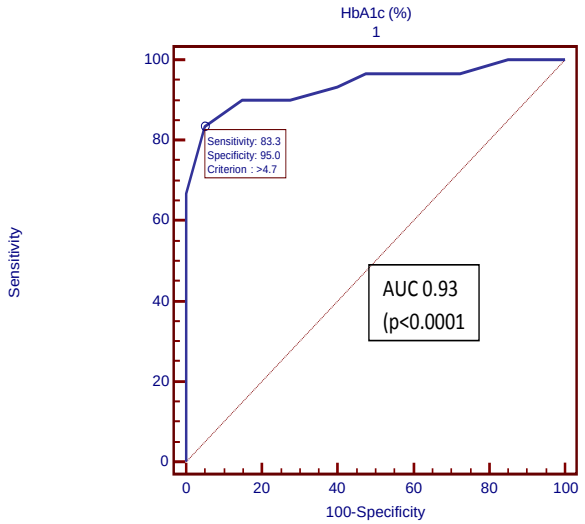

Figure 2: Receiver operating characteristic (ROC) curve of the HbA1c% for prediction of cardiomyopathy

Citation: Abdel-Azeem M. El-Mazary; Madiha Sayed; Hazem Ali; Waleed Abdel Hameed; Nancy Tawadros. "Cord Blood Hemoglobin A1c and MCP-1 as Predictors for Cardiomyopathy in Infants of Mothers with Gestational Diabetes". Annals of Neonatology Journal, 1(2), 2019: 59-71. doi: 10.21608/anj.2019.45815