Cord Blood Adropin as an early predictor for fetal Growth and Cardiac Complications in Infants of Diabetic Mothers

Original Article

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ABSTRACT

Background: Adropin is a polypeptide involved in energy homeostasis, insulin response, and endothelial function, significantly impacting fetal development. Numerous studies suggest that it regulates fetal growth.

Aim of the Work: The current study sought to investigate cord blood adropin levels in infants of diabetic mothers and to correlate them with fetal growth, and cardiac complications.

Patients and Methods: Thirty infants of mothers with diabetes mellitus (IDM) and thirty infants whose mothers did not have diabetes were enrolled in this comparative case-control study. Adropin levels were assayed by non-competitive enzyme-linked immunosorbent assay in the cord blood of all enrolled neonates. Birth weight and ponderal index were recorded. Echocardiographic measurements were conducted in the first three days of life.

Results: Adropin levels were significantly lower in infants of diabetic mothers [median (IQR): 80.25 (55.5 – 99) pg/ml] compared to infants of non-diabetic mothers [median (IQR): 718.25 (487-944)]. Growth-related metrics (including birth weight and ponderal index) and echocardiographic measurements of left ventricular end-systolic dimensions were negatively correlated with adropin levels.

Conclusion: Cord blood adropin levels can be used as a foreteller of fetal growth and cardiac complications in infants of diabetic mothers.

Key Words: Diabetes Mellitus, echocardiography, marker, newborn.

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INTRODUCTION

Diabetes mellitus (DM) is a chronic systemic metabolic disorder marked by increased insulin resistance. It is a microvascular disorder that can affect individuals of all ages, from fetal development to late adulthood. Diabetes during pregnancy may either emerge for the first time during gestation and resolve or persist postpartum (gestational DM) or could be preexisting before pregnancy (pregestational DM). The prevalence of impaired glucose tolerance during pregnancy varies and is influenced by the overall incidence of diabetes in the general population[1].

Diabetes mellitus causes low-grade chronic inflammation in maternal and fetal bodies. Besides increasing the risk of maternal complications, it may also cause fetal and neonatal complications. These complications include congenital malformations, perinatal asphyxia, respiratory distress, macrosomia, and metabolic complications[2,3].

Adropin is a relatively recently investigated protein that can be used as a marker for inflammation. Adropin is thought to play a pivotal role in regulating placental development and function. It improves the placenta's metabolic efficiency by controlling glucose transport and enhancing insulin sensitivity, ensuring optimal nutrient transfer from the mother to the fetus. This regulation is essential for maintaining a proper balance between maternal and fetal nutrient needs, helping to prevent conditions such as fetal growth restriction or macrosomia[4]. Previous research on patients with obstructive sleep apnea has shown an inverse relationship between adropin and inflammatory markers such as hs CRP and interleukin-6, suggesting that adropin plays an anti-inflammatory role[5].

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Interestingly, research suggests that adropin deficiency contributes to the onset and progression of chronic diseases, including diabetes mellitus. This finding has been supported by some studies that reported reduced circulating adropin levels in adults with type 2 diabetes, individuals with liver disease, and children with type 1 diabetes mellitus[6-8].

Based on current literature, we hypothesized that changes in adropin levels in the cord blood of infants of diabetic mothers might indicate fetal exposure to inflammation during the antenatal period, which can have adverse effects on fetal growth and cardiac complications.

AIM OF THE WORK

This study sought to assess cord blood adropin levels in infants born to diabetic mothers compared to those born to non-diabetic mothers and to examine their correlation with fetal growth, glucose homeostasis, and cardiac complications.

PATIENTS AND METHODS

This comparative case-control study was performed at the Neonatal Intensive Care Unit (NICU) of Ain Shams University Hospitals, over six months; between May 2024 and November 2024.

The study included 60 full-term and near-term neonates; divided into two groups: 1) Group I (IDM group): included thirty neonates born to mothers with DM. 2) Group II (Control group): included thirty neonates born to non-diabetic mothers.

Inclusion criteria:

1. Full-term and near-term neonates (≥ 34 gestational weeks). The gestational age of the infants was determined by the date of the mother's last menstrual period or the modified Ballard score.

2. Neonates born to mothers with DM (HbA1c levels in mothers were more than or equal to 6.5%)

3. Neonates born to mothers without DM (HbA1c levels in mothers were below 5.7%)

Exclusion Criteria:

1. Neonates with significant congenital anomalies or chromosomal abnormalities.

2. Neonates diagnosed with birth asphyxia.

3. Neonates with a history of prenatal infections or maternal sepsis risk factors (e.g., chorioamnionitis).

4. Neonates whose mothers smoked during pregnancy.

5. Mothers who experienced pregnancy complications such as preeclampsia or hypertension.

6. Neonates born to mothers with metabolic or endocrine disorders, including thyroid disease or obesity.

All participants were subjected to the following:

1. Gestational age, sex, birth weight, ponderal index [birth weight (g) x 100/Height (cm3)], Apgar score at 1st and 5th minutes, and the modes of delivery were recorded.

2. Maternal Health Information was recorded:

- In the cases group: the time of diagnosis (whether it was pre-existing or gestational diabetes) and the type of diabetes treatment administered during pregnancy (such as insulin or oral hypoglycemics) were recorded

- In the control group: the recorded data was to ensure the absence of confounding conditions that could impact neonatal outcomes.

3. Vital signs (heart and respiratory rates) were assessed for neonates immediately after birth and every six hours for the first 24 hours.

4. Glucose and C-reactive protein (CRP) serum levels are measured for neonates from peripheral shortly after birth using the Roche/Hitachi Cobas C501 System (Roche Diagnostics International Ltd., Switzerland).

5. Cord blood adropin level measurement: 2 ml of cord blood was withdrawn immediately after birth into plain tubes with gel for serum separation. Samples were left to clot at room temperature for 30 minutes. Samples were centrifuged at 2000-3000 rpm for 20 minutes. The serum was separated, aliquoted, and stored at -20 °C until the assay. The assay used a commercially available sandwich (non-competitive) enzyme-linked immunosorbent assay kit from Sino Gene Clon Biotech Company, China; Catalog No: SG-11594.

6. Echocardiographic measurements were conducted following the guidance of the American Society of Echocardiography in the first three days of life. The procedure included measurement of the systolic and diastolic interventricular septal thickness (IVSs, IVSd), systolic and diastolic

left ventricular posterior wall thickness (LVPWs, LVPWd), in addition to end-systolic and diastolic left ventricular dimensions (LVESD, LVEDD).

ETHICAL CONSIDERATION

The study was carried out following approval of the local ethics committee, faculty of Medicine, Ain-shams University, and obtaining written/oral informed consent from the legal guardians of enrolled cases and controls (FMASU MS242/2024)

STATISTICAL ANALYSIS

Data were gathered, reviewed, coded, and analyzed by the Statistical Package for Social Science (IBM SPSS) (IBM Corp. Released 2020. IBM SPSS Statistics for Windows, Version 27.0. Armonk, NY: IBM Corp). Quantitative data were expressed as means and standard deviations when parametric. Nonparametric quantitative data were reported as medians and interquartile ranges (IQR). The qualitative variables were expressed as numbers and percentages. Group comparisons for qualitative data were conducted using the Chi-square test and/or Fisher exact test when the expected count in any cell was less than 5. The comparison between two independent groups with quantitative data was done using independent t-test for those with parametric distribution, and Mann-Whitney test for non-parametric data. Spearman correlation coefficients were used to assess the correlation between two quantitative parameters. The confidence interval was 95%, and the accepted error margin was 5%. So, the p-value significance was regarded as the following: *P-value* > 0.05: Non-significant (NS), *P-value* < 0.05: Significant (S), *P-value* < 0.01: Highly Significant (HS).

RESULTS

Our study included sixty infants. They were categorized into two groups: a) Group I: (The IDM group), and b) Group II : (The control group). The demographic data and clinical characteristics of the two groups are presented in (Table 1). The comparison between both groups regarding the vital signs (heart rate and respiratory rate) is shown in (Table 2). Both tables revealed no significant difference between the two groups as regards the studied parameters.

Table 1: Comparison between the IDM group and the control group regarding demographic data and Clinical Characteristics.

		Group I (IDM)	Group II (Control)	Test value	*P-value*	Sig.
		No. = 30	No. = 30			
Sex	Female	11 (36.7%)	13 (43.3%)	0.278*	0.598	NS
	Male	19 (63.3%)	17 (56.7%)			
Gestational Age (weeks)	Median (IQR)	36(35 – 37)	37(36 – 38)	-1.144≠	0.253	NS
	Range	35 – 42	35 – 41			
Mode of delivery	Cesarean section	30 (100.0%)	30 (100.0%)	-	-	-
Apgar score 1st min.	Median (IQR)	6(5 – 7)	6(6 – 7)	-0.302≠	0.763	NS
	Range	2 – 8	3 – 9			
Apgar score 5th min.	Median (IQR)	8 (8 – 8)	8 (8 – 8)	-1.175≠	0.240	NS
	Range	7 – 9	7 – 9			

*: Chi-square test; ≠: Mann-Whitney test

Table 2: Comparison between the IDM group and the control group regarding vital data measured at different times.

		Group I (IDM)	Group II (Control)	Test value	*P-value*	Sig.
		No. = 30	No. = 30			
Heart rate (beat/min.)						
At birth	Mean ± SD	141.07 ± 5.48	140.67 ± 4.3	-0.315•	0.754	NS
	Range	130 – 150	135 – 150			
6 hours after birth	Mean ± SD	140.5 ± 6.61	140.17 ± 4.45	-0.229•	0.820	NS
	Range	130 – 150	135 – 150			
12 hours after birth	Mean ± SD	139.83 ± 6.09	142 ± 5.51	1.446•	0.154	NS
	Range	130 – 150	135 – 150			
18 hours after birth	Mean ± SD	142.33 ± 4.69	141 ± 5.15	-1.049•	0.299	NS
	Range	130 – 150	135 – 150			
24 hours after birth	Mean ± SD	139.17 ± 4.93	141.67 ± 4.97	1.956•	0.055	NS
	Range	130 – 150	135 – 150			
Respiratory rate (min)						
At birth per min	Mean ± SD	42.3 ± 1.78	42.2 ± 1.97	-0.206•	0.838	NS
	Range	40 – 45	40 – 45			
6 hours after birth per min	Mean ± SD	41.87 ± 1.55	42.27 ± 2.03	0.857•	0.395	NS
	Range	40 – 45	40 – 45			
12 hours after birth per min	Mean ± SD	41.8 ± 1.69	42.3 ± 2.05	1.030•	0.307	NS
	Range	39 – 45	40 – 45			
18 hours after birth per min	Mean ± SD	42.23 ± 1.76	42.57 ± 1.96	0.694•	0.490	NS
	Range	40 – 45	40 – 45			
24 hours after birth per min	Mean ± SD	42.5 ± 1.83	42.53 ± 2.01	0.067•	0.947	NS
	Range	40 – 45	40 – 45			

•: Independent t-test

On the other hand, A statistically significant increase in birth weight (kg) and ponderal index (gm/cm3) was recorded in the IDM group compared to the control group (Table 3).

Table 3: Comparison between the IDM group and the control group regarding growth-related metrics.

		Group I (IDM)	Group II (Control)	Test value	*P-value*	Sig.
		No. = 30	No. = 30			
Birth weight	Mean ± SD	3.42 ± 0.62	2.65 ± 0.46	-5.420•	0.000	HS
(kg)	Range	2.5 – 4.6	1.9 – 3.7			
Ponderal index	Mean ± SD	3.41 ± 0.23	2.34 ± 0.31	-15.081•	0.000	HS
(g/cm3)	Range	3 – 3.9	1.8 – 2.8			

•: Independent t-test

The laboratory parameters of both groups are presented in Table 4. The IDM group had significantly lower glucose levels at birth. Adropin levels in cord blood were markedly lower in the IDM group compared to the controls. However, CRP levels showed no significant difference between both groups (Table 4)

Table 4: Comparison between the IDM group and the control group regarding laboratory parameters.

		Group I (IDM)	Group II (Control)	Test value	*P-value*	Sig.
Glucose at birth (mg/dl)	Mean ± SD	56.13 ± 16.56	82.03 ± 10.76	7.184•	0.000	HS
	Range	25 – 97	42 – 99			
Serum Adropin level (pg/ml)	Median (IQR)	80.25 (55.5 – 99)	718.25 (487 – 944)	6.654≠	0.001	HS
	Range	11 – 127	250 – 1350			
CRP (gm/L)	Mean ± SD	2.02±0.22	1.96±0.24	1.049•	0.299	NS
	Range					

•: Independent t-test, ≠: Mann-Whitney test

There was no significant difference in serum adropin levels among IDMs, regardless of whether their mothers had controlled (HbA1C ≤ 7) or uncontrolled diabetes (HbA1C >7) (Table 5)

Table 5: Comparison between serum adropin levels in IDM of both controlled and uncontrolled mothers.

Serum Adropin level (pg/ml)	Controlled (HbA 1C ≤ 7) no=20	Uncontrolled (HbA 1C > 7) no=10	Test value	*P-value*	Sig.
Median (IQR) Range	72.25 (40.25- 101.5) 11-127	91.5 (58- 93.5) 35-112	0.550	0.582	NS

In the correlation study, adropin levels showed a significant negative correlation with birth weight, Ponderal index, and echocardiographic finding (LVES) dimensions (Table 6).

Table 6: Correlation between cord blood adropin and clinical, laboratory and echocardiographic findings in infants of diabetic mothers.

(IDM) group	Serum Adropin level (pg/ml)	
	Test value (r)	*P-value*
GA (Bellard score)	0.305	0.101
Birth weight (kg)	-0.557**	0.001
Ponderal index (gm/cm3) (2.2-3.2)	-0.481**	0.007
Mother HbA1C	-0.023	0.906
Blood glucose at birth	0.189	0.318
Echo (IVSs) cm (0.26-0.82)	0.040	0.835
Echo (IVSd) cm (0.24-0.52)	-0.089	0.638
Echo (LVPWs) cm (0.35-0.79)	0.129	0.497
Echo (LVPWd) cm (0.26-0.48)	0.170	0.370
Echo (LVES) dimension (cm) (1.02-1.52)	-0.493**	0.006
Echo (LVED) dimension (cm) (1.65-2.33)	0.346	0.061

**: Spearman rank correlation coefficient

DISCUSSION

In the current study, significant variations were noted in growth-related metrics. The IDM group displayed higher birth weight and ponderal index. In the same contest, ***Deshpande-Joshi et al.*** (2024) recorded that maternal obesity and glucose intolerance during pregnancy contribute to increased neonatal birth weights and a higher rate of macrosomia[9]. These findings highlight the impact of maternal hyperglycemia and metabolic dysregulation on excessive fetal growth and adiposity.

Inaddition, Our results demonstrated that neonates in the IDM group exhibited significantly lower blood glucose levels than their counterparts in the control group. Continuous glucose monitoring studies have provided detailed insights into the glycemic patterns of IDMs. The results of ***Stewart et al.,*** (2019) study agreed with our findings. It revealed persistent hypoglycemia within the first 72 hours of neonates born to mothers with type 1 diabetes, with 25% of neonates spending over half of the first day with glucose levels below 47 mg/dL[10]. This risk is largely attributed to fetal hyperinsulinemia caused by chronic maternal hyperglycemia during pregnancy. This highlights the need for early and frequent glucose monitoring in IDMs to ensure timely interventions.

Moreover, our data demonstrated significant negative correlations between adropin levels and growth metrics in the IDM group. These findings indicate that elevated adropin levels may play a regulatory function in mitigating excessive fetal growth, which is often a common outcome of maternal hyperglycemia during pregnancy. Our findings align with a study done by ***Alzoughool et al.*** (2021); who studied serum adropin levels in type 2 diabetes mellitus patients and identified a significant negative correlation between adropin levels and anthropometric obesity indices like body mass index (BMI) and waist circumference[11]. This supports our conclusion that adropin may have a regulatory role in the growth and metabolic adaptations of neonates exposed to maternal diabetes.

Furthermore, our study monitored the vital parameters of neonates, including the heart and respiratory rates at various time points. The analysis revealed that these parameters remained stable over time in both groups, with no observed statistically significant differences between the infants of the diabetic mothers group and the control group. In the same contest, Thaseen and Veeraiah (2021) performed a study on neonates born to diabetic and non-diabetic mothers. Their findings revealed no significant differences in heart rate or blood pressure between the two groups, suggesting that maternal diabetes under control does not adversely affect neonatal vital parameters[12]. In contrast, some studies identified significant cardiovascular and metabolic abnormalities, including fluctuations in heart rate and blood pressure among neonates born to diabetic mothers. These disruptions were attributed to maternal hyperglycemia and inadequate control during pregnancy, which could affect neonatal autonomic stability[13].

Moreover, our study showed that adropin levels don't significantly correlate with blood glucose levels in the IDM group. Although IDMs exhibited lower serum glucose levels overall, these differences were not directly associated with serum adropin concentrations. This lack of correlation suggests that while adropin is decreased in neonates born to diabetic mothers, it may not serve as a direct indicator of glucose metabolism or glycemic control in the early neonatal period.

A significant negative correlation was detected in our study between serum adropin levels and the left ventricular end-systolic dimension. This finding suggests that decreased adropin levels may play a role in the impairment of left ventricular systolic function in neonates exposed to maternal diabetes. Our study aligns with previous research highlighting the impact of maternal diabetes on neonatal cardiac health and metabolic markers. For instance, ***Tejaswi et al.*** (2020) reported significant myocardial hypertrophy in neonates of diabetic mothers, particularly those with poorly controlled diabetes. The study linked myocardial hypertrophy with suboptimal maternal glycemic control, reinforcing our findings of structural cardiac changes influenced by maternal diabetes. These findings further emphasize the importance of maternal glycemic control in mitigating neonatal cardiac changes[14].

CONCLUSION

Our study detected alteration in cord blood adropin levels in IDM, which may be attributed to the inflammatory state itself or an impaired inflammatory response caused by DM. Based on these findings, serum adropin may serve as a promising indicator of meta-inflammation and a potential predictor of DM-related complications, particularly neonatal macrosomia, and hypertrophic cardiomyopathy, which are significant complications of pregnancy with DM.

DECLARATIONS

CONSENT FOR PUBLICATION

Not applicable.

AVAILABILITY OF DATA AND MATERIALS

The datasets utilized and/or analyzed in this study can be obtained from the corresponding author upon reasonable request.

COMPETING INTEREST

The authors declare no competing interests.

FUNDING

This study is self-funded.

AUTHORS' CONTRIBUTIONS

Soha Khafagy, Nancy Abo Shady, and Mohamed Metwally analyzed and interpreted neonatal and maternal clinical data and reviewed the selection criteria of all enrolled neonates. Miral Aref analyzed and interpreted neonatal cardiac investigations. Dina Ghanem and Madona Youssef performed the laboratory investigations and interpreted their results. Dina Ghanem and Madona Zaki played a significant role in writing the manuscript. All authors reviewed and approved the final version.

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Not applicable

LIST OF ABBREVIATIONS

IDM: Infants of Diabetic Mothers

DM: Diabetes mellitus

NICU: Neonatal Intensive Care Unit

CRP: C-reactive protein

IQR: Interquartile Range

IVSs: Systolic Interventricular Septal Thickness

IVSd: Diastolic Interventricular Septal Thickness

LVPWs: Systolic Left Ventricular Posterior Wall Thickness

LVPWd: Diastolic Left Ventricular Posterior Wall Thickness

LVESD: End-Systolic Left Ventricular Dimension

LVEDD: Diastolic Left Ventricular Dimension

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الأدروبين في دم الحبل السري كمؤشر مبكر على نمو الجنين والمضاعفات القلبية لدى حديثي الولادة من أمهات مصابات بداء السكري

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الخلفية: الأدروبين هو ببتيد متعدد يشارك في توازن الطاقة، واستجابة الأنسولين، ووظيفة البطانة الوعائية، مما يؤثر بشكل كبير على نمو الجنين. تشير العديد من الدراسات إلى أنه ينظم نمو الجنين.

هدف الدراسة: هدفت هذه الدراسة إلى البحث في مستويات الأدروبين في دم الحبل السري لدى حديثي الولادة من أمهات مصابات بداء السكري، وربطها بنمو الجنين والمضاعفات القلبية.

المرضى والطرق: شملت هذه الدراسة المقارنة من نوع الحالات والشواهد ثلاثين رضيعًا لأمهات مصابات بداء السكري (IDM) وثلاثين رضيعًا لأمهات غير مصابات بداء السكري. تم قياس مستويات الأدروبين باستخدام اختبار الامتصاص المناعي المرتبط بالإنزيم غير التنافسي (ELISA) في دم الحبل السري لجميع حديثي الولادة المشاركين. تم تسجيل وزن الولادة ومعامل بونديرال، كما تم إجراء قياسات تخطيط صدى القلب خلال الأيام الثلاثة الأولى من الحياة.

النتائج: كانت مستويات الأدروبين أقل بشكل ملحوظ لدى حديثي الولادة من أمهات مصابات بداء السكري [الوسيط (المدى الربيعي): 80.25 (55.5 – 99) بيكوغرام/مل] مقارنة بحديثي الولادة من أمهات غير مصابات بالسكري [الوسيط (المدى الربيعي): 718.25 (944-487)]. كما لوحظ وجود ارتباط سلبي بين مستويات الأدروبين ومقاييس النمو (بما في ذلك وزن الولادة ومعامل بونديرال) وقياسات تخطيط صدى القلب لأبعاد نهاية الانقباض البطيني الأيسر.

الاستنتاج: يمكن استخدام مستويات الأدروبين في دم الحبل السري كمؤشر على نمو الجنين والمضاعفات القلبية لدى حديثي الولادة من أمهات مصابات بداء السكري.