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Original Article

Evaluation of the relationship between neonatal serum asprosin levels and anthropometric measurements in newborns of mothers with and without gestational diabetes mellitus

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ABSTRACT

Background. Asprosin is a newly identified adipokine that is expressed in the placenta. Its production is increased in women with gestational diabetes mellitus (GDM), and it is a factor related to insulin resistance. This study aimed to determine whether neonatal serum asprosin levels are associated with anthropometric characteristics of newborns born to mothers with and without GDM.

Methods. This study included 51 newborns of mothers with GDM (insulin-treated or diet-treated) and 55 control newborns with their mothers. In newborns, anthropometric parameters were measured, and the concentrations of asprosin were detected by ELISA. Maternal blood glucose levels, body weight, and length were measured and body mass index (BMI) was calculated.

Results. Serum asprosin levels were significantly higher and linked to a higher risk in the newborns of mothers with GDM compared with those of the control newborns (170.3 [132.6] vs. 91.4 [68.7] ng/mL, $p < 0.001$). Serum asprosin levels were negatively correlated with blood glucose concentrations ($r = -0.282$, $p = 0.045$) in the newborns of mothers with GDM and significantly positively correlated with birth weight ($r = 0.315$, $p = 0.019$) in the control newborns. Newborn serum asprosin levels were positively correlated with the glucose levels ($r = 0.264$, $p = 0.006$) of all mothers. In addition, newborns born to an insulin-treated mother with GDM had significantly higher birth weight and length than newborns born to a diet-treated mother with GDM (3262.9 vs. 3137 g, $p = 0.032$, and 49.7 vs. 49.2 cm, $p = 0.05$). Although asprosin levels were higher in newborns of mothers treated with insulin, these differences were not statistically significant. Mothers with GDM had high blood glucose levels ($p = 0.032$).

Conclusions. Serum levels of asprosin are increased and negatively correlated with glucose concentrations in newborns of mothers with GDM. Asprosin could be used as an early biomarker in newborns of GDM mothers.

Key words: asprosin, newborn, gestational diabetes mellitus.

The asymptomatic condition known as gestational diabetes mellitus (GDM) is defined by carbohydrate intolerance that transforms into diabetes during pregnancy despite normal glucose metabolism prior to pregnancy.[1] There are many short- and long-term consequences for the mother and fetus when maternal diabetes occurs during pregnancy. GDM has a number of causes, including the occurrence of insulin resistance as a result of the anti-insulin actions of placental hormones and the increase in maternal adipose tissue during pregnancy.[2] Babies born to mothers with GDM are more likely to acquire type II diabetes later in life and to be overweight or obese at an early age.[3]

Two exons (exons 65 and 66) of the fibrillin-1 gene (*FBN1*) encode the newly identified,

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glucogenic adipokine asprosin, which is produced and released by white adipose tissue during fasting. Asprosin plays a complex role in the central nervous system (CNS), peripheral tissues, and organs.[4] Through a multitude of signaling channels, asprosin significantly influences things like hunger, insulin resistance, glucose metabolism, and cell death.[5] Asprosin is crucial in the treatment of metabolic disorders such as insulin resistance, type 2 diabetes, and polycystic ovary syndrome.[6,7] Asprosin concentrations are considerably greater in type 2 diabetic women, and asprosin and insulin resistance are positively correlated in polycystic ovarian syndrome patients.[8] Patients with glucose dysregulation have significantly higher asprosin concentrations, which are linked to a number of clinical indicators of lipid and glucose metabolic disorders.[9] Zhong et al.[10] found that the placenta expresses asprosin and that GDM pregnant women have higher levels. Baykus et al.[11] reported that infants with intrauterine growth restriction had the lowest asprosin concentrations while pre-eclampsia, gestational diabetes, and fetal macrosomia were associated with higher salivary and blood asprosin levels. Although there are studies on maternal and neonatal asprosin levels, including gestational diabetes, pre-eclampsia, and fetal growth retardation[11,12], the underlying mechanisms for its formation are not fully known. Therefore, the goal of this study was to investigate the relationship between neonatal serum asprosin levels and anthropometric features in babies delivered in Turkish to women with and without gestational diabetes.

Material and Methods

Study population

This prospective study was conducted in the Neonatology Unit of Kütahya Health Sciences University from December 2020 to December 2021. The study was approved by the Local Ethics Committee of Kütahya Health Sciences University (date: November 25, 2020; no. 2020-07/04). Parental consent was obtained before blood samples were taken. The mothers were diagnosed with gestational diabetes by an oral glucose tolerance test (OGTT) performed between 24 and 28 gestational weeks, and their 51 newborns of mothers with gestational diabetes (30 diet-treated vs. 21 insulin-treated) were included in the study. The mothers, in whom the OGTT results were normal, and their 55 newborns (37–41 weeks of gestation, both groups) were accepted into the control group. Maternal blood glucose levels, body weight and length were measured and body mass index (BMI) was calculated by dividing weight by the square of height (kg/m^2).

Gestational diabetes mellitus was diagnosed according to the American Diabetes Association's suggested criteria (fasting blood sugar of ≥ 92 < 126 mg/dL at the time of the first examination and the presence of at least one abnormal result (fasting ≥92 mg/dL, 1st hour ≥180 mg/dL, 2nd hour ≥153 mg/dL) in a 75 g oral glucose tolerance test after 24–28 weeks).[13] It was noted that glucose monitoring, diet, and exercise were recommended to pregnant women diagnosed as having GDM, and insulin therapy was initiated by consulting endocrinology for pregnant women whose targeted glucose values could not be determined.

After birth, the gestational age at delivery, and weight, length, and head circumference of the newborns were recorded and ponderal index (PI) was calculated using the formula: PI = [weight (g) x 100] ÷ [length (cm)]^3. The body weight of newborns was measured using electronic scales sensitive to 5 g. Length measurements were taken (head part fixed, foot part movable) using a length measurement board.

Blood samples were taken from newborns of mothers with and without GDM to measure glucose levels. Blood glucose assessments of newborns were made according to the American Academy of Pediatrics (AAP) criteria.[14] Since the newborns of mothers with GDM are at risk, blood glucose measurement was repeated 30 minutes after early feeding. If the blood glucose levels of newborns born to mothers with GDM

were normal at 12 hours of screening in large for gestational age (LGA) babies and at 24 hours in small for gestational age (SGA) babies, the screening was terminated.

Blood samples obtained from newborns to measure asprosin levels were placed in non-heparinized tubes and promptly centrifuged at 1,000 g for 10 min. The serum samples were then kept at −20 °C until analysis. Asprosin measurement in serum was performed with the Human Asprosin ELISA measurement kit (Bioassay Technology Laboratory, Catalogue no: E4095Hu, Shanghai, China). Absorbance reading was done on a Chromate 4300 brand ELISA Reader device (Awareness Technology, Inc., Palm City, USA). Asprosin test results were given as ng/mL, with an assay range of 0.5 to 100 ng/mL and a sensitivity of 0.23 ng/mL.

Babies of pregnant mothers with similar BMI values were included in the study. Mothers with pregestational diabetes or a history of chronic disease and infants with maternal clinical conditions such as parathyroid, a bone, kidney, or gastrointestinal disorder or with congenital anomalies were excluded.

Statistical analysis

All analyses were performed using SPSS version 20.0 software (SPSS Inc., Chicago, USA). The Kolmogorov–Smirnov test was performed to check the normality of distributions. The independent samples t test was used for normally distributed variables such as birth weight, body weight, length, and BMI, and are presented as mean ± standard deviation (SD). The Mann–Whitney U test was used for non-normally distributed variables such as gestational age, birth length, head circumference, PI, glucose, and asprosin, and are presented as median and interquartile range. Categorical data like sex are presented as number and percentage using chi-square ($\chi 2$) test. To evaluate the variables related with mothers and their newborns were analyzed using binary and multivariate logistic regression analysis to evaluate the odds ratios (OR) and 95% confidence intervals (CIs). Hosmer-Lemeshow test was used to check the model fitness. Pearson's correlation was used to correlate serum asprosin with birth weight, body weight, length, BMI. Spearman's correlation was used to correlate serum asprosin levels with gestational age, birth length, head circumference, PI, and glucose. Linear regression analysis was used to evaluate the relationship between neonatal serum asprosin and other variables. P values ≤ 0.05 were considered statistically significant.

Results

The newborn and mother characteristics are summarized in Table I. In all, 106 newborns were consecutively recruited, including 51 newborns of mothers with GDM and 55 newborns of non-GDM mothers (control), and matched for gestational age [37.0 (36.0–38.0) vs. 38.0 (37.0–39.0) weeks; $p < 0.001$] and sex (28 males/23 females vs. 26/29; $p = 0.432$). There was no significant difference in birth weight, birth length, BMI, head circumference, PI, or blood glucose levels between the newborn groups. Factors associated with mothers and their newborns were determined by logistic regression analysis and the results are shown in Table II. Serum asprosin levels were significantly higher in the newborns of mothers with GDM than in the control ($p < 0.001$), and the OR was approximately 1.08 (95% CI 1.04–1.12, $p < 0.001$). In newborns of mothers with non-GDM there was increased gestational age compared to the newborns of mothers with GDM ($p < 0.001$), and OR value was 0.61 (95% CI 0.46–0.82, $p = 0.001$). In addition, mothers with GDM had a significantly higher glucose concentration compared to non-GDM mothers [90.0 (81.0–103.0) vs. 98.0 (88.0–110.0) mg/dl; $p = 0.032$], and the OR was 1.02 (95% CI 1.00–1.04, $p = 0.041$) as shown in Table I and Table II.

In our study, the GDM group was divided into two subgroups including those treated with diet (n=30) and insulin (n=21). As depicted in Table III, neonates born to an insulin-treated mother with GDM had a significantly higher

Table I. Demographic and clinical characteristics of mothers and their newborns.

	Control newborns (n = 55)	Newborns of mothers with GDM (n = 51)	*p*
Male / Female (n)	26 / 29	28 / 23	0.432
Gestational age (weeks, range)	38 (37 - 39)	37 (36 - 38)	< 0.001*
Birth weight (g)	3194 ± 321	3189 ± 567	0.958
Birth length (cm)	50 (49 - 51)	49 (48 - 50)	0.245
BMI (kg/m^2)	12.9 ± 0.95	12.9 ± 1.69	0.797
Head circumference (cm)	34.1 (34.0 - 35.0)	34.5 (34.0 - 35.0)	0.868
PI (kg/m^3)	2.60 (2.50 - 2.70)	2.55 (2.40 - 2.86)	0.766
Glucose (mg/dL)	78.0 (64.0 - 89.0)	71.0 (62.0 - 87.0)	0.171
Asprosin (ng/mL)	91.4 (68.7 - 114.3)	170.3 (132.6 – 236.9)	< 0.001*
	Control mothers (n = 55)	Mothers with GDM (n = 51)	*p*
Glucose (mg/dL)	90.0 (81.0 - 103.0)	98.0 (88.0 - 110.0)	0.032*
Body weight (kg)	77.2 ± 13.7	76.9 ± 12.0	0.769
Length (cm)	161.8 ± 6.8	160.5 ± 5.5	0.105
BMI (kg/m^2)	29.4 ± 4.5	29.8 ± 4.3	0.872
Delivery method			
Vaginal (n, %)	25 (59.5)	17 (40.5)	0.202
Cesarean (n, %)	30 (46.9)	34 (53.1)	
Treatment Diet/Insulin (n, %)	-	30 (58.8) / 21 (41.2)	-

Birth weight, body weight, length, BMI were described as mean ± standard deviation and determined by independent Student t-test. Other parameters were described as median and interquartile range and determined by Mann-Whitney U test. Proportion n (%) was determined by chi-square (χ2).
*p ≤ 0.05 is considered signifcant. BMI: body mass index, GDM: gestational diabetes mellitus, PI: ponderal index.

Table II. Logistic regression analysis determining the factors associated with mothers and their newborns.

Newborns	Univariate logistic regression		Multivariate logistic regression	
	OR (95% CI)	p	OR (95% CI)	p
Male/Female (n)	0.73 (0.34 – 1.58)	0.433	-	-
Gestational age (weeks)	0.61 (0.46 – 0.82)	0.001*	0.59 (0.30 – 1.14)	0.120
Birth weight (g)	1.00 (0.99 – 1.00)	0.957	-	-
Birth length (cm)	0.91 (0.73 – 1.14)	0.432	-	-
BMI (kg/m^2)	1.03 (0.78 – 1.37)	0.795	-	-
Head circumference (cm)	0.94 (0.64 – 1.38)	0.775	-	-
PI (kg/m^3)	1.78 (0.42 – 7.62)	0.431	-	-
Glucose (mg/dL)	0.98 (0.96 – 1.00)	0.096	1.01 (0.97 – 1.05)	0.498
Asprosin (ng/mL)	1.08 (1.04 – 1.12)	<0.001*	1.08 (1.048 – 1.13)	<0.001*
Mothers	Univariate logistic regression		Multivariate logistic regression	
	OR (95% CI)	p		
Glucose (mg/dL)	1.02 (1.00 – 1.04)	0.041*	1.01 (0.98 – 1.05)	0.377
Body weight (kg)	0.99 (0.96 – 1.02)	0.890	-	-
Length (cm)	0.96 (0.90 – 1.02)	0.273	-	-
BMI (kg/m^2)	1.02 (0.93 – 1.11)	0.654	-	-
Method of Delivery				
Vaginal (n, %)	1.67 (0.75 – 3.66)	0.204	-	-
Cesarean (n, %)				
Treatment Diet/Insulin (n, %)	-	-	-	-

Logistic regression analysis was performed to evaluate the odds ratios (OR) and 95% confidence intervals (CIs).
*p ≤ 0.05 is considered signifcant. BMI: body mass index, GDM: gestational diabetes mellitus, PI: ponderal index.

birth weight and birth length compared with neonates born to a diet-treated mother (p = 0.032, p ≤ 0.05, respectively). Although the newborn asprosin levels tended to be higher in the insulin group, these differences were not statistically significant (p = 0.108) (Table III). In addition, glucose levels were greater in the insulin-treated GDM mothers compared to the diet-treated mothers (p ≤ 0.05).

Correlation analyzes between anthropometric variables and serum asprosin concentrations in the newborns and mothers are shown in Table IV. For all newborn groups, serum asprosin levels correlated negatively with gestational age (r = −0.272; p = 0.005) and glucose concentration (r = −0.267; p = 0.006). In addition, a positive correlation between serum levels of asprosin, birth weight (r = 0.187; p = 0.05), BMI (r = 0.247; p = 0.011), and the mother's glucose concentration (r = 0.264; p = 0.006) was found in all groups. There was a significant positive correlation between asprosin, birth weight, and BMI in the newborns of the non-GDM mothers (r = 0.315; p = 0.019 and r = 0.291; p = 0.031, respectively). In newborns of mothers without GDM, both gestational age and birth weight were high, and there was a positive correlation with each other (r = 0.415, p < 0.001). In the newborns of mothers with GDM, serum asprosin levels correlated negatively with blood glucose (r = −0.282; p = 0.045) and positively with BMI (r = −0.336; p = 0.016). There was no relationship between serum asprosin levels and birth length, head circumference or PI in newborns (Table IV). The BMI of mothers with GDM was negatively correlated with newborn glucose levels (r = −0.307; p = 0.02), while birth weight (r = 0.416; p = 0.002) and birth length (r = 0.365; p = 0.008) were positively correlated. After multivariate linear regression analysis, neonatal serum asprosin levels continued to be negatively related to gestational age and glucose level of newborns and positively related to maternal glucose level in the all group (Table IV).

Informed consent was obtained from all individual participants' legal guardians included in the study.

Discussion

In this study, we investigated the relationship between blood asprosin concentrations and anthropometric characteristics in newborns born to women with gestational diabetes in the Turkish population. Asprosin, an orexigenic hormone that increases hepatic glucose production, is a possible therapeutic target for the treatment of both obesity and diabetes.[4,15] Patients with impaired glucose regulation have significantly higher asprosin levels, which are correlated with several clinical markers of lipid and glucose metabolic disorders.[16] Asprosin, a

Table III. Clinical outcomes of newborns of mothers with GDM treated with diet and insulin.

Newborns	Diet (n=30)	Insulin (n=21)	p
Male / Female (n)	17 / 13	11 / 10	0.493
Gestational age (weeks)	37 (36 - 38)	37 (36 - 38)	0.410
Birth weight (g)	3137 ± 473	3263 ± 686	0.032*
Birth length (cm)	49.2 ± 1.7	49.7 ± 2.3	0.05*
BMI (kg/m^2)	12.9 ± 1.51	13.0 ± 1.95	0.703
Head circumference (cm)	34.8 (34-35)	34 (33 - 35)	0.260
PI (kg/m^3)	2.64 ± 0.32	2.61 ± 0.34	0.604
Glucose (mg/dL)	73.8 ± 17.1	73.2 ± 18.5	0.511
Asprosin (ng/mL)	161.4 (131.4 – 227.5)	194.8 (149.2 - 252.9)	0.108

Birth weight, birth length, BMI, PI, glucose were described as mean ± standard deviation and determined by independent Student t-test. Other parameters were described as median and interquartile range and determined by Mann-Whitney U test. Proportion n (%) was determined by chi-square (χ2).
*p ≤ 0.05 is considered signifcant. BMI: body mass index, GDM: gestational diabetes mellitus, PI: ponderal index.

Table IV. Correlation between neonatal serum asprosin concentrations and various parameters in mothers and their newborns.

Newborns	Control newborns (n = 55)				Newborns of mothers with GDM (n = 51)				All newborns (n = 106)			
	Univariate correlation analysis		Multivariate regression analysis		Univariate correlation analysis		Multivariate regression analysis		Univariate correlation analysis		Multivariate regression analysis	
	r	p	β	p	r	p	β	p	r	p	β	p
Gestational age (weeks)	0.095	0.488	-	-	−0.151	0.290	-	-	−0.272	0.005*	-0.369	<0.001*
Birth weight (g)	0.315	0.019*	0.226	0.302	0.257	0.069	-	-	0.187	0.05*	0.079	0.687
Birth length (cm)	0.154	0.261	-	-	−0.045	0.756	-	-	−0.036	0.713	-	-
BMI (kg/m^2)	0.291	0.031*	0.111	0.612	0.336	0.016*	0.294	0.036*	0.247	0.011*	0.239	0.203
Head circumference (cm)	0.173	0.207	-	-	−0.011	0.938	-	-	0.050	0.611	-	-
PI (kg/m^3)	0.143	0.297	-	-	0.255	0.071	-	-	0.135	0.166	-	-
Glucose (mg/dL)	−0.223	0.101	-	-	−0.282	0.045*	-0.201	0.146	−0.267	0.006*	-0.267	0.003*
Mothers	Control mothers (n = 55)				Mothers with GDM (n = 51)				All mothers (n = 106)			
	Univariate correlation analysis		Multivariate regression analysis		Univariate correlation analysis		Multivariate regression analysis		Univariate correlation analysis		Multivariate regression analysis	
	r	*p*	β	*p*	r	*p*	β	*p*	r	*p*	β	*p*
Glucose (mg/dL)	0.083	0.547	-	-	0.210	0.138	-	-	0.264	0.006*	0.193	0.047*
Body weight (kg)	−0.084	0.543	-	-	0.030	0.834	-	-	-0.018	0.851	-	-
Length (cm)	−0.163	0.233	-	-	0.148	0.300	-	-	−0.062	0.530	-	-
BMI (kg/m^2)	0.007	0.957	-	-	-0.036	0.803	-	-	0.018	0.857	-	-

Pearson correlation analysis was used for normally distributed variables as birth weight, body weight, length, BMI. Spearman correlation analysis was used for skewness distribution as gestational age, birth length, head circumference, PI, and glucose. Linear regression analysis was used to evaluate the relationship between serum asprosin and other variables. *$p \leq 0.05$ is considered signifcant. BMI: body mass index, GDM: gestational diabetes mellitus, PI: ponderal index.

novel factor associated with insulin resistance, may play a role in the development of GDM.[10] There is currently little information available about asprosin levels in pregnant women with GDM and their newborns. The findings of Baykus et al.[11] indicated that venous and arterial cord blood asprosin levels in newborns of GDM pregnant women were higher. Birth weights of infants and asprosin levels in arterial and venous cord blood were positively correlated in women with gestational diabetes. Zhong et al.[10] demonstrated that asprosin levels were elevated in the umbilical cord of newborns from GDM mothers; neonatal cord asprosin levels showed a positive correlation with birth weight. In this study, serum concentrations of asprosin were significantly positively correlated with birth weighs and BMI, and negatively correlated with gestational age and blood glucose levels in all newborn groups. Serum concentrations of asprosin were also significantly positively correlated with birth weight and BMI in newborns of non-GDM mothers. In addition, levels of serum asprosin were significantly higher in newborns of mothers with GDM and were correlated negatively with blood glucose concentration and positively with BMI. In newborns born to mothers with GDM, increased serum asprosin levels may be an important risk factor.

Baykus et al.[11] and Zhong et al.[10] demonstrated that asprosin levels are elevated in the plasma of pregnant women with GDM. Further, asprosin levels are positively associated with age in pregnant women but not with maternal BMI.[10] In our study, mothers with GDM had high blood glucose levels. We were unable to look at maternal serum insulin, hemoglobin A1c (HbA1c), and asprosin levels in this study. Therefore, we could not examine the relationship between maternal blood sugar and asprosin level. But, there was a positive correlation between the asprosin levels of newborns and maternal blood glucose levels. The reason for the increase in serum asprosin levels in newborns of mothers with GDM may be due to the increase in hepatic glucose production due to changes in maternal insulin sensitivity during pregnancy.

GDM is a developing health problem worldwide affecting up to 14% of pregnancies according to the diagnostic criteria and demographics analyzed.[13-17] Pre-pregnancy maternal obesity, excessive weight gain during pregnancy and gestational diabetes are the main causes of pathological pregnancy conditions. A newborn's energy metabolism becomes compromised due to insulin resistance, high blood glucose, and hormonal changes.[18] In our study, gestational age was significantly lower among newborns of mothers with GDM than among newborns of non-GDM mothers. There was no significant difference in birth weights, birth length, BMI, head circumference, or PI between the newborn groups. Other studies have not shown differences in birth measurements between neonates exposed to GDM and those not.[19,20] According to a study by Bayoumi et al.[21], there is no statistically significant difference between babies born to healthy non-diabetic women and babies born to women with GDM in terms of length or head circumference. Contrarily, Baptiste-Roberts et al.[22] demonstrated that mothers with GDM delivered infants with higher birth weight than mothers without diabetes. In addition, Sletner et al.[23] found that compared to offspring of non-GDM mothers, offspring of GDM mothers were smaller in the middle of pregnancy but developed more rapidly until delivery. This disparity may be attributable to the diverse ethnic backgrounds of the participants.

GDM is linked to complications in both mothers and newborns.[24] Neonatal hypoglycaemia is one such complication; it generally develops in the first 24 hours after birth as newborns go through their metabolic transition during the first few days of life.[25,26] Rarely, prolonged or recurrent hypoglycemia can have serious and long-lasting neurological health consequences, and babies with GDM mothers are especially at risk.[27] Neonatal hypoglycaemia and congenital anomalies are more prevalent in infants born to women with GDM.[28,29] Among neonates whose

mothers had GDM, neonatal hypoglycemia and hyperbilirubinemia are more common.[28] The likelihood of neonatal hypoglycemia in diabetic mothers is the consequence of a combination of risk factors, including maternal blood glucose levels, maternal treatment, and birth weight, according to studies.[30-33] In our study, although the blood glucose levels of babies born to mothers with GDM were lower than those of non-diabetic mothers, the difference was not significant. In addition, babies of mothers with GDM treated with insulin or diet showed no difference in blood glucose levels. The nonsignificance of these results may be due to the low number of pregnant women included in the study.

In this study, glucose levels were higher in insulin-treated mothers with GDM compared with diet-treated mothers with GDM. Compared to neonates born to diet-treated mothers, neonates born to insulin-treated mothers with GDM had considerably higher birth weight and birth length. In research by Koning et al.[34] newborns in the insulin group had lower birth weights and gestational ages at birth than those in the diet group. We found that although not statistically significant, serum asprosin levels tended to be higher in neonates born to insulin-treated mothers with GDM. Placental asprosin levels are correlated with maternal insulin levels and rise after starting insulin therapy in GDM patients, according to Hoffman et al.[35] There are no literature data showing serum asprosin levels in neonates born to insulin-treated mothers with GDM.

The current study has the following limitations: 1) asprosin levels were only tested in the neonates' peripheral blood; 2) asprosin levels in the peripheral blood of the mothers were not analyzed; and 3) the number of newborns was relatively low because this was a single-center clinical study involving the Turkish population. The relationship between anthropometric measurements and asprosin in neonates during GDM as well as the causes for increased asprosin release are unknown. It is advised that our findings be replicated in larger, more ethnically diverse populations in order to support the validity of our conclusions.

In the newborns of mothers with GDM, asprosin concentration was considerably higher and had a negative correlation with blood glucose levels. Higher serum asprosin levels may raise the risk of short- and long-term negative health outcomes, such as neonatal and obstetric problems during delivery and obesity and diabetes later in life for these babies. Therefore, elevated asprosin levels may be an important risk factor for newborns born to mothers with GDM, independent of anthropometric measurements.

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Ethical approval

This study was started following the decision of the Local Ethics Committee of Kütahya Health Sciences University (date: November 25, 2020, and no. 2020- 07/04). Informed consent was obtained from all individual participants' legal guardians included in the study.

Author contribution

The authors confirm contribution to the paper as follows: study conception and design: EEY, RA; data collection: EEY; analysis and interpretation of results: RA, EEY; draft manuscript preparation: EEY, RA. All authors reviewed the results and approved the final version of the manuscript.

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Conflict of interest

The authors declare that there is no conflict of interest.

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