Annals of Clinical and Analytical Medicine
Original Research

Investigation of serum asprosin level in gestational diabetes mellitus and examination of neonatal outcome

Asprosin in GDM and neonatal outcomes

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Abstract

Aim: Our aim in this study is to investigate serum asprosin levels in Gestational Diabetes Mellitus (GDM) patients and to examine the neonatal outcomes of these patients.

Material and Methods: Pregnant women between the ages of 24/0–28/6 weeks who came to our clinic between June 2021 and February 2022 for an oral glucose tolerance test (OGTT) were included in the study. The patients were divided into two groups based on the findings of the OGTT: the GDM group (n = 52) and the control group (n = 31). All patients' serum values for asprosin, triglycerides, HDL, LDL, total cholesterol, and insulin were examined. To measure the difference in asprosin level between trimesters, it was studied for the second time in all patients between 32 and 36 weeks.

Results: The GDM group's elevated HbA1c was statistically significant when analyzed alongside the control group. The newborns in the GDM group were measured as longer than the infants in the control group. The infants in the GDM group had a lower 5-minute APGAR score. The first aprosin value was substantially lower in the GDM group than in the control group. There was no variation in the second asprosin value among the groups. There was no discernible link between serum asprosin levels and the outcomes of newborns.

Discussion: No correlation was observed between serum asprosin levels, GDM and neonatal outcomes. It would be beneficial to conduct more extensive studies on this subject.

Keywords

Asprosin, Gestational Diabetes Mellitus, Neonatal Outcome

DOI: 10.4328/ACAM.22112 Received: 2024-01-16 Accepted: 2024-03-05 Published Online: 2024-03-18 Printed: 2024-04-01 Ann Clin Anal Med 2024;15(4):287-292
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This study was approved by the Ethics Committee of Necmettin Erbakan University (Date: 2021-06-18, No: 2021/3300)

Introduction

During the second half of pregnancy, catabolism takes over due to the action of placental hormones. The hormones progesterone, estrogen, prolactin, cortisol, and especially human placental lactogen, create a diabetogenic environment by showing an anti-insulin effect, and maternal blood glucose levels are kept at high levels both during fasting and satiety to meet the increasing need of the fetus.

The fibrillin-1 gene in adipose tissue encodes the adipokine asprosin. Romere gave the initial description of it in 2016 [1]. Asprosin has been shown to have a role in several complex processes, such as glucose metabolism, the emergence of insulin resistance (IR) in peripheral tissues and organs, and the apoptosis of brain cells [2, 3]. The asprosin level increases with food intake and is directly proportional to body weight. Its function in the body is to increase hepatic glucose secretion [1]. Studies on humans and mice have demonstrated in the literature that asprosin is linked to obesity, polycystic ovarian syndrome (PCOS), IR, and cardiovascular diseases (CVD) [2, 4].

The illness known as diabetes mellitus (DM) is brought on by a disruption in the metabolism of proteins, fats, and carbohydrates as a result of either insufficient or nonexistent pancreatic insulin secretion [5]. A carbohydrate intolerance problem identified as gestational diabetes mellitus (GDM) develops during pregnancy and typically goes away after the pregnancy is over [6]. Pregnancy-related problems are widespread, with GDM being one of them. The main maternal effects of GDM are hyperglycemia, hypertension and preeclampsia. Macrosomia, perinatal mortality, newborn respiratory issues, and metabolic abnormalities (hypoglycemia, hypocalcemia, and hyperbilirubinemia) are the primary impacts of gestational diabetes mellitus on fetuses and infants.

The most commonly used procedure in clinics is the 75-gram oral glucose tolerance test (OGTT), which is approved by the World Health Organization (WHO) but there are additional methods. This test is carried out between weeks 24 and 28 of pregnancy and can be used for both screening and diagnosis.

Diagnosis of GDM is very important aspect of maternal, fetus and infant health, and to ensure strict blood sugar regulation with diet or insulin. Asprosin is believed to have a function in this regulation and could, thus, serve as a substitute for OGTT in the diagnosis of GDM. The purpose of this research was to look at the asprosin level in the serum and assess the newborn outcomes of GDM patients.

Material and Methods

This prospective, cross-sectional research investigation was conducted out in the obstetrics and gynecology clinic of a tertiary hospital in Turkey between June 2021 and February 2022.

The study's inclusion requirements were singleton pregnancies with normal first-trimester fasting blood sugar levels, ages 18–45, gestational weeks 24/0–28/6, consenting to an OGTT, and willing participation in the research. Ages <18 or >45, neurocognitive illnesses preventing them from answering the consent form, gestational ages ≤24/0 or ≥28/6 weeks, multiple pregnancies, any other diseases, or incapacity to withstand the OGTT were the exclusion criteria for the study.

Based on prior comparable research, the sample size was computed to yield a standard effect size of 0.82 for a minimum of 23 patients per group, with a five percentage point margin of mistake and 80% power [7]. Two groups of patients were created: The GDM group (n = 52) and the control group (n = 31). The WHO and IADPSG (International Association of Diabetes and Pregnancy Study Group) suggested the use of OGTT o 75 grams as a diagnostic and screening test. The test was conducted, and the threshold values established by both organizations were applied. Accordingly, in the diagnosis of GDM, the threshold values used were blood glucose after eight hours of fasting ≥92 mg/dL; after 75 g OGTT first hour blood glucose ≥180 mg/dL, and second hour ≥153 mg/dL [8]. Any time any one of these amounts was elevated, GDM was diagnosed.

Age, prior pregnancies, history of gestational diabetes, and gestational week were among the sociodemographic data analyzed for the two patient groups. Anthropometric data included length, weight, BMI, waist and hip circumference, and waist-hip ratio (WHR). Laboratory values included HDL, LDL, total cholesterol, triglyceride, HbA1c, insulin, hemoglobin, hematocrit, and platelets. Finally, neonatal data included delivery type, birth week, gender, height-weight, head circumference, fetal distress, APGAR 1–5th minute, cord pH, neonatal intensive care admission, and poor neonatal outcome (stillbirth, hypothermia, hypoglycemia, respiratory distress syndrome). Using the blood remaining from the blood sample taken in the normal follow-up examination of pregnant women, the asprosin level was examined twice using the sandwich ELISA kit (Bioassay Technology Laboratory company) with ELx800 Absorbance Microplate Reader (Biotek, Winooski, VT, USA) at gestational week 24/0-28/6 and than after 32/0 weeks.

Statistical Analysis

SPSS vn 28.0 software was used to statistically analyze the study's data. The terms mean ±, standard deviation, median, lowest and greatest values or numbers (n), and percentage (%) were used for descriptive statistics. The Kolmogorov-Smirnov test was used to determine if the variables conformed to a normal distribution. The quantitative independent data was analyzed using the Mann-Whitney U test. When the requirements of the Chi-square test were not satisfied, the Fischer test was utilized in place of the Chi-square test for the examination of qualitative independent data. It was determined that a value of ($p<0.05$) was statistically significant.

Etihical Approval

This study was approved by the Ethics Committee of Necmettin Erbakan University (Date: 2021-06-18, No: 2021/3300).

Results

Two patient groups were evaluated: the control group (n = 31) and the GDM group (n = 52). Table 1-2 provides demographic data, serum indicators, and the average neonatal outcome values for each pregnant research participant. Age, gravida, parity, and the number of living children were all considerably greater in the GDM group ($p<0.05$). In terms of the other sociodemographic data, there was no significant variation between the groups ($p>0.05$). When compared to the control group, the GDM group's WBC and HbA1c values were considerably greater ($p<0.05$). The GDM group's initial aprosin value was significantly ($p<0.05$)

Table 1. General characteristics of all the patients and the neonatal outcomes

		Min-Max			Median	Mean±SD/n-%		
Age (years)		18.0	-	40.0	28.0	27.7	±	5.9
Pre-Pregnancy Weight (kg)		44.0	-	126	68.0	70.2	±	16.0
Weight at Study baseline (kg)		55.0	-	128	75.0	78.1	±	14.8
Waist Circumference (cm)		82.0	-	141	98.0	99.2	±	10.2
Hip Circumference (cm)		91.0	-	150	110	112	±	11.9
Gravida		1.0	-	9.0	3.0	3.2	±	1.8
Parity		0.0	-	8.0	1.0	1.8	±	1.7
Normal OGTT / Diet / Insulin	Normal OGTT					31		37.3%
	Diet					39		47.0%
	Insulin					13		15.7%
Insulin		2.3	-	290	21.3	34.7	±	40.2
LDL		42.0	-	228	146	143	±	39.2
HbA1c		4.6	-	8.0	5.3	5.4	±	0.53
1 st Asprosin measurement		8.9	-	366	18.5	40.8	±	70.1
2 nd Asprosin measurement		9.1	-	747	20.0	54.4	±	110
Neonatal Data		**Min-Max**			**Median**	**Mean±SD/n-%**		
Birth Week		34.0	-	41.0	39.0	38.5	±	1.5
Lenght (cm)		45.0	-	57.0	50.5	50.6	±	2.2
Weight (gr)		2200	-	4405	3230	3240	±	419
Head circumference (cm)		30.0	-	37.0	35.0	34.7	±	1.3
APGAR 1 Min		5.0	-	9.0	9.0	8.4	±	0.7
APGAR 5 Min		8.0	-	10.0	10.0	9.5	±	0.6
Intensive Care Hospitalization						2		2.4%
RDS and CPR						0		0.0%
Neonatal Tachypnea						2		2.4%
Hypoglycemia						2		2.4%

Normal OGTT; normal oral glucose tolerance test, HbA1c: Hemoglobin A1C, LDL: Low-density lipoprotein, RDS; respiratory distress syndrome, CPR; Cardiopulmonary Resuscitation

Table 2. Comparison of the demographic data and serum values of the groups

	Control Group (n=31)				GDM Group (n=52)				P	
	Mean±SD/n-%			Median	Mean±SD/n-%			Median		
Age (years)	25.6	±	5.4	24.0	29.0	±	5.9	30.0	0.013	m
Weight (kg)	75.9	±	14.1	75.0	79.4	±	15.2	75.5	0.349	m
Lenght (cm)	157.9	±	5.2	158.0	159.2	±	5.6	158.0	0.517	m
Waist Circumference (cm)	97.6	±	8.9	98.0	100.1	±	10.9	98.5	0.429	m
Hip Circumference (Cm)	109.9	±	11.5	110.0	113.9	±	12.0	112.0	0.162	m
Gravida	2.6	±	1.6	2.0	3.5	±	1.9	3.0	0.040	m
Number of Parities	1.13	±	1.26	1.00	2.13	±	1.82	2.00	0.008	m
Number of Abortions	0.5	±	1.0	0.0	0.3	±	0.7	0.0	0.795	m
Number of Living Children	1.2	±	1.2	1.0	2.2	±	2.0	2.0	0.012	m
Gestational week when OGTT performed	27.4	±	1.1	28.0	27.0	±	1.4	28.0	0.318	m
WBC	10.9	±	2.5	10.5	9.3	±	2.2	8.8	0.004	m
Insulin	37.7	±	54.9	22.3	33.0	±	28.6	20.1	0.608	m
Total Cholesterol	225.1	±	43.7	220.0	227.6	±	45.8	231.0	0.836	m
LDL	142.9	±	36.2	146.0	142.6	±	41.3	146.0	0.888	m
HDL	64.5	±	15.3	61.0	59.6	±	13.1	56.0	0.119	m
HbA1c	5.1	±	0.3	5.2	5.5	±	0.6	5.3	0.001	m
Triglyceride	212.1	±	63.5	196.0	231.0	±	75.8	218.0	0.194	m
1. Asprosin*	57.1	±	93.1	20.2	30.1	±	47.7	16.1	0.033	m
2. Asprosin**	79.0	±	158.1	19.8	37.7	±	55.8	20.0	0.956	m

m Mann-Whitney U test
GDM: Gestational diabetes mellitus, OGGT; Oral glucose tolerance test, HbA1c: Hemoglobin A1C, LDL: Low-density lipoprotein, HDL: High density-lipoprotein, OGGT: Oral glucose tolerance test, WBC; white blood cell, *Asprosin 1; measured at 24-28 weeks of gestation. **Asprosin 2; measured at 32-36 weeks of gestation.

Table 3. Comparison of the neonatal results of the groups

	Control Group				GDM Group				p	
	Mean±SD/n-%			Median	Mean±SD/n-%			Median		
Birth Week	38.6	±	1.9	39.0	38.5	±	1.3	39.0	0.247	m
Lenght (cm)	50.0	±	2.1	50.0	51.0	±	2.2	51.0	0.014	m
Weight (gr)	3165	±	421	3100	3286	±	416	3390	0.141	m
Head circumference (cm)	34.5	±	1.3	35.0	34.8	±	1.3	35.0	0.338	m
Fetal Distress	3		9.7%		9		17.3%		0.339	X^2
Primary C/S	3		9.7%		8		15.4%		0.458	X^2
Cord pH	7.3	±	0.05	7.3	7.3	±	0.0	7.3	0.962	m
APGAR 1 Min	8.6	±	0.6	9.0	8.3	±	0.8	8.0	0.076	m
APGAR 5 Min	9.7	±	0.47	10.0	9.4	±	0.60	9.0	0.011	m
Intensive Care Hospitalization	0		0.0%		2		3.8%		0.262	X^2
RDS	0		0.0%		0		0.0%		1.000	X^2
Neonatal tachypnea	0		0.0%		2		3.8%		0.526	X^2
Hypoglycemia	0		0.0%		2		3.8%		0.526	X^2
CPR	0		0.0%		0		0.0%		1.000	X^2

X^2 Chi-square / m Mann-Whitney U test
GDM: Gestational diabetes mellitus, C/S; Cesarean Section, RDS; Respiratory distress syndrome, CPR; Cardiopulmonary Resuscitation

lower than the control group's. The second aprosin value did not significantly differ across the groups (p>0.05). Insulin levels or any other lipid markers did not differ significantly across the groups (p> 0.05). (Table 2) Compared to the control group, the infant length in the GDM group was significantly (p<0.05) lengthier. There was no discernible difference between the groups when other neonatal demographic data were taken into account (p>0.05). The GDM group's APGAR 5-minute value was significantly lower (p<0.05) than that of the control group. There was no statistically significant difference observed between the groups with respect to the cord pH, APGAR 1 min score, and poor neonatal outcomes (p>0.05). (Table 3).

Discussion

GDM is the most common systemic disease in pregnancy and although the true incidence is not known, reported prevalence varies between 1-14% [9]. Over the past 20 years, there has been a rise in the prevalence of GDM, primarily due to older mothers and an increase in childhood obesity [10]. Complications can be avoided with appropriate follow-up and treatment.

When fasting, the liver produces glucose due to the secretion of asprosin, which affects blood sugar regulation and insulin resistance (IR) [5]. Asprosin also has the effect of preventing pancreatic beta cells from secreting insulin [11]. It has been demonstrated that asprosin affects intricate processes in the brain, spinal cord, tissues in the peripheral region, and organs, including hunger, glucose metabolism, IR, and cell destruction [1, 6].

Obesity, personal and family medical history, and advanced maternal age all raise the potential of GDM, even if its exact cause is unknown [12]. The likelihood of having GDM rises with increasing gestational age, gravida, and parity numbers [5,13]. In keeping with previous research, the GDM group in this study outperformed the control group in terms of birth rates, age, gravida number, parity number, parity ratio, amount of live children, and ratio of living children (p<0.05).

In contrast to prior studies, there was no discernible difference between the research groups in terms of triglycerides, LDL, HDL, and total cholesterol when the lipid profile was assessed in the second trimester in GDM and control groups. This could be because diet controls blood sugar levels in the majority of GDM pregnant women. However, there are various results in the literature regarding this [14, 15]. Feig et al. reported that pregnant women with GDM and healthy pregnant women had greater insulin levels than non-pregnant women [16]. In the current study, pregnant women without GDM and those in good health were compared. Despite considerably higher insulin levels in the control group, no statistically noteworthy variance was observed (p>0.05).

In 2019, Baykus et al. Reported that plasma asprosin levels were higher in pregnant women with GDM, severe preeclampsia and macrosomic fetuses between 37-39 weeks of pregnancy compared to healthy pregnant women [17]. In the current investigation, asprosin levels were shown to be higher in the second and third trimesters among healthy pregnant women than in the group with gestational diabetes mellitus. There was no statistically significant difference (p>0.05) between the groups in the second asprosin measurement, although there was a statistically significant difference (p<0.05) in the first asprosin measurement. The lower asprosin level in the GDM group in the current study may be explained by the sample size limitations and the fact that a higher percentage of GDM patients were treated with food rather than insulin. The Baykus et al study did not separate the GDM patients into diet- or insulin-regulated categories. If the GDM patients had been separated into two groups or if there had been more diet-regulated GDM patients, perhaps asprosin would have been determined at a lower level in the GDM group of that study. Furthermore, it's unclear if the asprosin level remained significant because it wasn't examined for two trimesters in the Baykus et al study.

In another study by Zhong et al serum asprosin values were examined in pregnant women with GDM at 3 different times (18-20w, 24-28w and in the prenatal period). There was no appreciable change between the groups' asprosin values

measured between 24-28w; however, the GDM group's results for other asprosin [7]. In the current study, the asprosin value was not high in the GDM group. Differences between the studies could also be attributed to the use of different brand kits and there is no specific cut-off value in the asprosin kit.

Upon combining all of these findings, it is evident that additional research with a larger sample size and comparison studies using OGTT—the gold standard of diagnostic evaluation—is required to ascertain whether asprosin may be utilized in the diagnosis of GDM. There may be a negative correlation, not a positive one, between asprosin and GDM. Asprosin is not commonly utilized, nevertheless, because there is a lack of a standard for the asprosin value ranges and discrepancies in the measurement and confidence intervals of the kits available on the market. These issues make it difficult to do additional research.

Previous studies have found significantly increased HbA1c levels in GDM patients [18,19]. These investigations support the findings that the GDM group in the current study had a higher HbA1c ($p<0.05$). According to Fagninou et al., pregnant GDM patients had considerably higher WBC levels than the control group [20]. Conversely, the current study's findings indicated that the GDM group's WBC value was lower than the control group's ($p<0.05$).

Fuka et al. looked at the neonatal outcomes of GDM-affected pregnant women and discovered that the infants' 5-min APGAR score was lower in GDM patients [21]. The APGAR score of infants whose pregnancies were affected by GDM was similarly shown to be low in another study that looked at maternal and neonatal outcomes [22]. Regarding the first-minute APGAR score, there was no discernible difference ($p>0.05$) between the GDM group and the control group in this investigation. On the other hand, the GDM group's fifth-minute APGAR score was considerably lower ($p<0.05$). These findings are consistent with the information found in the literature, leading one to believe that babies of GDM patients should have close follow-up because the general health of these infants may worsen over the postnatal period. The association between asprosin level during pregnancy and neonatal outcomes could not be explored in this investigation because, in contrast to other studies, the asprosin level was not found to be high in the GDM group. Consequently, it was believed that the existence of GDM was the cause of the poor neonatal outcomes. Relationship building with asprosin was not achievable.

This study has some limitations. Among these was the fact that asprosin levels in fetal cord blood were not investigated, nor were asprosin levels in non-pregnant individuals tested. In addition, a group of patients were excluded from the study because they could not be followed up regularly or gave birth in another hospital. Finally, when the diagnosis of GDM was made, the first diet followed by insulin therapy made it difficult to recruit patients. However, this study has some strengths. One of its strongest points is that this is the first study, to our knowledge, to examine the effects of asprosin on neonatal outcomes. Another is that it is one of the few studies in the literature examining the change in asprosin levels between trimesters.

Conclusion

According to the study's findings, there was no discernible difference in the groups' serum asprosin levels between the GDM patients and the control group, and the pregnancies' neonatal outcomes were comparable. However, it would be beneficial to conduct more extensive studies on this subject.

Scientific Responsibility Statement

The authors declare that they are responsible for the article's scientific content including study design, data collection, analysis and interpretation, writing, some of the main line, or all of the preparation and scientific review of the contents and approval of the final version of the article.

Animal and Human Rights Statement

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or compareable ethical standards.

Funding: *None*

Conflict of Interest

The authors declare that there is no conflict of interest.

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How to cite this article:
Emre Uysal, Feyzanur Incesu Cintesun, Oguzhan Gunenc, Ummugulsum Can. Investigation of serum asprosin level in gestational diabetes mellitus and examination of neonatal outcome. Ann Clin Anal Med 2024;15(4):287-292

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