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Maternal Serum Visfatin, Adiponectin, Human Placental Lactogen, and Insulin as Potential Biomarkers of Fetal Macrosomia in Gestational Diabetes Mellitus

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ABSTRACT: Gestational diabetes mellitus (GDM) is linked to metabolic and endocrine changes that may contribute to the development of fetal macrosomia and excessive fetal growth. The present study aimed to evaluate some maternal metabolic biomarkers; visfatin, adiponectin, human placental lactogen (HPL), and insulin concentrations, as well as their correlation and diagnostic performance, in pregnancies with fetal macrosomia induced by gestational diabetes.

In the third trimester (28–36 weeks of gestation), a case-control study was conducted on 90 pregnant women. The study included 60 women with GDM who were carrying a macrosomic fetus and 30 healthy pregnant women who maintained normal fetal growth. enzyme-linked immunosorbent assay (ELISA) was employed to determine the concentrations of visfatin, adiponectin, HPL, and insulin in the maternal serum. Pearson's correlation coefficient, receiver operating characteristic (ROC) curve analysis, and independent-samples t-test were implemented in the statistical analyses. Maternal serum visfatin (3.136 ± 0.33 vs. 1.842 ± 0.27 ng/mL), HPL (475.61 ± 29.55 vs. 338.06 ± 19.21 ng/mL), and insulin (8.28 ± 0.22 vs. 8.12 ± 0.15 mU/L) concentrations were significantly higher in pregnancies with fetal macrosomia than in healthy controls, whereas adiponectin concentrations were significantly lower (9.03 ± 0.17 vs. 13.62 ± 0.60 ng/mL) ($P < 0.01$). In the macrosomia group, correlation analysis revealed substantial positive correlations between adiponectin and HPL, estimated fetal weight, and visfatin. The diagnostic performance of adiponectin was the highest (AUC = 0.744), followed by visfatin (AUC = 0.681), according to the ROC analysis. HPL exhibited limited diagnostic performance (AUC = 0.508). These findings suggest that adiponectin may be a promising biomarker for identifying pregnancies at increased risk of fetal macrosomia, and that maternal adipokines and placental hormones are closely associated with the metabolic disturbances underlying fetal macrosomia

1. INTRODUCTION

Gestational diabetes mellitus (GDM) is a metabolic disorder that commonly affects pregnancy and is distinguished by glucose intolerance that is initially identified during gestation[1]. Due to the increasing prevalence of maternal obesity, delayed childbearing, and sedentary lifestyles, the global prevalence of GDM has seen a significant increase in recent decades[2]. GDM is linked to a variety of adverse maternal and neonatal outcomes, such as fetal macrosomia, respiratory complications, neonatal hypoglycemia, cesarean delivery, and hypertensive disorders[3]. GDM continues to be a significant public health concern due

to its short- and long-term metabolic consequences for both mothers and their offspring, despite improvements in antenatal care[4].

One of the most common complications of gestational diabetes mellitus (GDM) is fetal macrosomia, which is typically defined by an abnormal intrauterine metabolic environment that leads to excessive fetal growth[5]. Maternal hyperglycemia induces hyperinsulinemia and fetal pancreatic β -cell hyperplasia by increasing the transport of glucose from the placenta to the fetus[6]. Because fetal insulin acts as a potent growth factor, an increase in fetal insulin secretion results in increased glycogen synthesis, lipid deposition, and protein synthesis, which ultimately results in excessive fetal growth and an increase in birth weight[7]. As a result, pregnancies that are complicated by fetal macrosomia are associated with a higher incidence of shoulder dystocia, birth trauma, cesarean delivery, and adverse neonatal metabolic outcomes[8].

Maternal metabolic adaptation during pregnancy is regulated by a complex interaction between adipokines and placental hormones, which affect insulin sensitivity, glucose homeostasis, and nutrient transport to the developing fetus[9]. Among these biomarkers, visfatin has been implicated in glucose metabolism and possesses insulin-mimetic and pro-inflammatory properties, whereas adiponectin enhances insulin sensitivity and exerts anti-inflammatory effects[10]. A peptide hormone secreted by the syncytiotrophoblast, human placental lactogen (HPL), promotes physiological insulin resistance during pregnancy ensuring an adequate supply of glucose and nutrients for fetal development[11]. Maternal insulin is a critical indicator of metabolic adaptation in pregnancies complicated by GDM, as it reflects the degree of insulin resistance and the function of pancreatic β -cells[12]. The development of fetal macrosomia and excessive fetal growth may be influenced by changes in these hormonal pathways[13].

Increasing evidence suggests that the pathophysiology of gestational diabetes mellitus and fetal overgrowth is significantly influenced by changes in maternal adipokines and placental hormones[7][14]. Visfatin, adiponectin, human placental lactogen (HPL), and insulin are among the most extensively investigated biomarkers due to their impact on glucose homeostasis, insulin sensitivity, placental function, and fetal growth regulation[15]. Maternal metabolic imbalance, excessive nutrient transfer across the placenta, and accelerated fetal growth may be influenced by dysregulation of these biomarkers[16]. Consequently, the assessment of their circulating maternal concentrations may offer valuable insights into the mechanisms that underlie fetal macrosomia and enhance comprehension of its metabolic foundation[17].

Thus, the current study aimed to evaluate maternal serum levels of insulin, visfatin, adiponectin, and human placental lactogen between healthy pregnancies and pregnancies affected by fetal macrosomia. The study also evaluated the correlations among these biomarkers and their diagnostic performance in predicting fetal macrosomia.

2.MATERIALS AND METHODS

2.1 Study Design and Participants

This case-control study was conducted at numerous obstetric clinics and maternity hospitals in Baghdad, Iraq, between November 2025 and April 2026. The study included a total of 90 pregnant women in the third trimester of pregnancy (28–36 weeks of gestation). The participants were divided into two groups: 60 pregnant women diagnosed with gestational diabetes mellitus (GDM) carrying a macrosomic fetus with weights (more than 4000 gm.) (case group), and 30 apparently healthy pregnant women without GDM carrying fetuses with estimated fetal weights (less than 4000 gm. (control group). Maternal demographic and clinical characteristics, such as maternal age, body mass index (BMI), gestational age, parity, previous history of macrosomia, mode of previous delivery, chronic diseases, fasting blood sugar (FBS), timing of GDM diagnosis, and treatment modality, were extracted from obstetric records and a structured questionnaire. Furthermore, fetal ultrasonography was used to evaluate the following parameters: biparietal diameter (BPD), head circumference (HC), abdominal circumference (AC), femur length (FL), estimated fetal weight (EFW), and amniotic fluid index (AFI).

2.2 Inclusion and Exclusion Criteria

This study was open to pregnant women aged 25–45 who were carrying singleton pregnancies between 28 and 36 weeks of gestation. The case group was composed of women who were diagnosed with gestational diabetes mellitus and were carrying a fetus with ultrasonographically diagnosed macrosomia. The control group consisted of pregnant women who were apparently healthy and within the same gestational age range, with normal fetal growth. The investigation excluded women with pre-existing type 1 or type 2 diabetes mellitus, chronic or infectious diseases during pregnancy, multiple pregnancies, unexplained

miscarriage, smoking, and a maternal age below 20 years. The Scientific and Ethical Committees of the Department of Biology, College of Science, University of Baghdad, formally conferred ethical approval. In compliance with the ethical standards for human subjects research, written informed consent was obtained from all participants prior to enrollment.

2.3 Blood Sample Collection

During routine antenatal visits, venous blood samples (5 mL) were obtained from all participants under aseptic conditions. Blood was transferred into plain tubes devoid of anticoagulant and allowed to clot at room temperature for 10–20 minutes. Serum was subsequently obtained by centrifuging the samples at 3000 rpm for 15–20 minutes. The serum that was separated was aliquoted into sterile Eppendorf tubes that were labeled and stored at -20°C until it was time for hormonal analysis. In order to ensure the reliability of the analytical results and preserve the stability of the samples, it was avoided to perform repeated freeze-thaw cycles.

2.4 Hormonal Assays

The manufacturer's instructions were followed for determining the concentrations of visfatin, adiponectin, human placental lactogen (HPL), and insulin in the maternal serum using commercially available sandwich enzyme-linked immunosorbent assay (ELISA) kits (SunLong Biotech, China). Using a microplate reader (BioBase, China), the absorbance was measured at 450 nm, and the concentrations of each analyte were determined from the corresponding standard calibration curves.

2.5 Statistical Analysis

Statistical analyses were performed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). The mean $\pm$ standard deviation (Mean $\pm$ SD) was used to express continuous variables. The Chi-square (χ^2) test was employed to compare categorical variables, while the independent-samples t-test was employed to compare the mean values between the case and control groups. The relationships between the biomarkers and clinical variables under investigation were assessed using Pearson's correlation coefficient (r). The diagnostic performance of the biomarkers under investigation was assessed by calculating the area under the curve (AUC), sensitivity, and specificity using receiver operating characteristic (ROC) curve analysis. A P value of less than 0.05 was regarded as statistically significant, while a P value of less than 0.01 indicated a highly significant difference.

3. RESULTS AND DISCUSSION

3.1 General characteristics of the study population

Table 1. General characteristics of pregnant women with fetal macrosomia and healthy controls.

Character		Pregnant women with macrosomic fetus (n=60)	Pregnant women with normal weight fetus (n=30)	P-value
Age (years)	<30	17 (28.33%)	12 (40.00%)	0.2337 NS
	31-35	34 (56.67%)	12 (40.00%)	0.0419 *
	>35	9 (15.00%)	6 (20.00%)	0.5022 NS
BMI (kg/m ²)	≤ 25	2 (3.33%)	20 (66.67%)	0.0001 **
	26-30	21 (35.00%)	10 (33.33%)	0.5627 NS
	>30	37 (61.67%)	0 (0.00%)	0.0001 **
Gestational age at delivery (GA) (week)	30-33	49 (81.67%)	8 (26.67%)	0.0001 **
	34-36	11 (18.33%)	22 (73.33%)	0.0001 **
Mode of delivery	Normal	27 (45.00%)	19 (63.33%)	0.0355 *
	C. Section	33 (55.00%)	11 (36.67%)	0.03351 *

Parity with macrosomia	Primipara (one macrosomia)	38 (63.33%)	-	0.0389 *
	Multipara (other than one macrosomia)	22 (36.67%)	-	
* (P<0.05), ** (P<0.01), NS: Non-Significant.				

The demographic and clinical characteristics of the study participants are summarized in Table 1. Body mass index (BMI), gestational age, parity, and mode of delivery differed significantly ($P<0.05$) between pregnant women with GDM complicated by fetal macrosomia and healthy pregnant women, maternal age was categorized into three groups, as shown in Table 1. Only the 31–35-year age group showed a significantly higher proportion in the macrosomia group than in the control group (56.67% vs. 40.00%, $P<0.05$), whereas no significant differences were observed in the <30 and >35 -year age groups.

The established association between maternal adiposity, insulin resistance, and excessive fetal growth is consistent with the higher BMI observed in the macrosomia group[18]. Impairment of glucose metabolism and increased placental nutrient transfer are the results of increased maternal adiposity, which in turn promotes fetal overgrowth[19]. Similarly, the increased frequency of cesarean delivery is indicative of the obstetric complications associated with fetal macrosomia, while the prolonged gestational age provides additional time for fetal growth[20]. The concept that maternal metabolic status significantly influences the development of fetal macrosomia is substantiated by these findings.

3.2 Maternal Serum Biomarkers

Table 2. Comparison of maternal serum visfatin, adiponectin, human placental lactogen, and insulin concentrations between women with GDM complicated by fetal macrosomia and healthy pregnant controls.

Parameters	Mean $\pm$ SD		P-value
	Pregnant women with macrosomic fetus (n=60)	Pregnant women with normal weight fetus (n=30)	
Visfatin (ng/mL)	3.136 $\pm$ 0.33	1.842 $\pm$ 0.27	0.0001 **
Adiponectin (ng/mL)	9.03 $\pm$ 0.17	13.62 $\pm$ 0.60	0.0001 **
HPL (ng/mL)	475.61 $\pm$ 29.55	338.06 $\pm$ 19.21	0.0001 **
Insulin (mU/L)	8.28 $\pm$ 0.22	8.12 $\pm$ 0.15	0.0005 **
** (P<0.01).			

The concentrations of visfatin, adiponectin, human placental lactogen (HPL), and insulin in the maternal serum of both study groups are summarized in Table 2. Serum visfatin concentrations were significantly higher in women with GDM carrying macrosomic fetuses (3.136 $\pm$ 0.33 ng/mL) than in healthy pregnant women (1.842 $\pm$ 0.27 ng/mL) ($P = 0.0001$). The elevated visfatin level may be a compensatory response to the chronic inflammatory state associated with GDM and maternal insulin resistance [21]. Adipose tissue and the placenta both produce visfatin, which is involved in the regulation of glucose homeostasis and inflammatory processes[14]. These results are in agreement with Jiang et al. [22], who reported elevated circulating visfatin concentrations in women with GDM, suggesting that metabolic disturbances during pregnancy may be linked to alterations in visfatin secretion.

Maternal serum adiponectin concentrations were significantly lower in women with GDM carrying macrosomic fetuses (9.03 $\pm$ 0.17 ng/mL) than in healthy pregnant women (13.62 $\pm$ 0.60 ng/mL) ($P = 0.0001$). Increased nutrient availability to the fetus and impaired maternal insulin sensitivity may result from reduced adiponectin, thereby fostering excessive fetal growth[7]. Adiponectin plays a critical function in the regulation of metabolic and inflammatory processes and the preservation of insulin sensitivity during pregnancy[23]. Gao et al. [24] reported an association between GDM, diminished adiponectin levels, and impaired insulin sensitivity, which is consistent with the current finding.

Maternal serum HPL concentrations were significantly higher in women with GDM carrying macrosomic fetuses (475.61 ± 29.55 ng/mL) than in healthy pregnant women (338.06 ± 19.21 ng/mL) ($P = 0.0001$). A significant placental hormone, HPL, reduces maternal insulin sensitivity and enhances the availability of glucose and other nutrients for fetal growth[11]. Consequently, the elevated concentration of HPL observed in the current study may be indicative of improved placental endocrine activity and heightened metabolic adaptation during late pregnancy[25].

Maternal serum insulin concentrations were significantly higher in women with GDM carrying macrosomic fetuses (8.28 ± 0.22 mU/L) than in healthy pregnant women (8.12 ± 0.15 mU/L) ($P = 0.0005$). The increased maternal insulin may be a compensatory response to the reduced insulin sensitivity that is usually associated with GDM[26]. During late pregnancy, the concurrent increase in insulin and HPL is indicative of the presence of progressive maternal insulin resistance and may increase the availability of nutrients for fetal growth[27].

The present study's observed altered levels of visfatin, adiponectin, HPL, and insulin collectively suggest that fetal macrosomia in pregnancies complicated by GDM is associated with disturbances in maternal metabolic regulation and placental endocrine function. The interaction between maternal insulin resistance, altered adipokine secretion, and placental hormonal adaptations may promote excessive fetal growth and increased nutrient availability[9].

3.3 Statistical Correlation Analysis

Table 3. Pearson's correlation coefficients between maternal serum hormonal biomarkers and selected maternal and fetal variables.

Variables	Visfatin (Macrosomia) r (P-value)	Visfatin (Control) r (P-value)	Adiponectin (Macrosomia) r (P-value)	Adiponectin (Control) r (P-value)
BMI	-0.10 (NS)	-0.02 (NS)	-0.04 (NS)	0.09 (NS)
Gestational age	0.08 (NS)	0.17 (NS)	0.13 (NS)	-0.07 (NS)
HPL	0.21 (NS)	0.02 (NS)	0.36 (0.0059)	0.28 (0.0486)
Insulin	0.15 (NS)	0.11 (NS)	0.18 (NS)	0.09 (NS)
Estimated fetal weight	0.13 (NS)	0.03 (NS)	0.27 (0.0498)	-0.23 (NS)
BPD	-0.06 (NS)	0.09 (NS)	0.03 (NS)	-0.34 (0.0451)
HC	-0.07 (NS)	0.08 (NS)	-0.002 (NS)	-0.32 (0.0476)
AC	-0.04 (NS)	0.007 (NS)	-0.04 (NS)	-0.01 (NS)
FL	-0.07 (NS)	0.24 (NS)	0.01 (NS)	-0.22 (NS)
AFI	0.16 (NS)	-0.06 (NS)	0.15 (NS)	-0.19 (NS)
Adiponectin	0.69 (0.0001)	0.02 (NS)	—	—

Abbreviations: BMI, body mass index; HPL, human placental lactogen; BPD, biparietal diameter; HC, head circumference; AC, abdominal circumference; FL, femur length; AFI, amniotic fluid index; NS, non-significant.

The relationships between maternal hormonal biomarkers and specific maternal and fetal characteristics are illustrated in Table 3. There was a strong positive correlation between adiponectin and maternal serum visfatin in the macrosomia group. Furthermore, adiponectin exhibited substantial positive correlations with HPL and estimated fetal weight, while the control group exhibited negative correlations with biparietal diameter and head circumference. Most of the remaining correlations were not statistically significant, indicating that maternal hormonal biomarkers are influenced by multiple maternal and placental factors rather than a single clinical parameter[28]. These results suggest that maternal hormonal changes in GDM-associated fetal macrosomia are part of a complex metabolic network that involves fetal growth and placental endocrine function, rather than solitary endocrine changes[29,30].

Table 4. Diagnostic performance of maternal serum hormonal biomarkers for predicting fetal macrosomia using ROC curve analysis.

Biomarker	Sensitivity (%)	Specificity (%)	AUC
Visfatin	57	71	0.681
Adiponectin	65	78	0.744
Human placental lactogen (HPL)	62	58	0.508

Abbreviations: ROC, receiver operating characteristic; AUC, area under the curve.

The diagnostic performance of maternal serum visfatin, adiponectin, and HPL was assessed using receiver operating characteristic (ROC) curve analysis, as summarized in Table 4. Adiponectin exhibited the highest diagnostic performance (AUC = 0.744), followed by visfatin (AUC = 0.681). HPL demonstrated limited diagnostic performance (AUC = 0.508). These results indicate that maternal adipokines, particularly adiponectin, may have a higher discriminatory value for identifying pregnancies complicated by fetal macrosomia than placental hormones alone.

4. CONCLUSIONS

In pregnancies complicated by gestational diabetes mellitus and fetal macrosomia, the present study revealed substantial changes in the concentrations of maternal serum visfatin, adiponectin, human placental lactogen, and insulin. Significant disturbances in maternal metabolic and placental endocrine function are suggested by elevated maternal serum visfatin, HPL, and insulin levels, as well as decreased adiponectin concentrations. The diagnostic performance of adiponectin was the highest among the biomarkers that were investigated for the identification of pregnancies that were complicated by fetal macrosomia. These results indicate that maternal hormonal biomarkers, particularly adipokines, may offer valuable insights into the pathophysiology of fetal macrosomia and could aid in the early identification of pregnancies at increased risk. Further large-scale studies are warranted to validate these findings and confirm the clinical applicability of these biomarkers.

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