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Assessment of Serum Omentin-1 Level in Iraqi women with Gestational Diabetes Mellitus

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Abstract

The condition known as gestational diabetes mellitus (GDM) is described as intolerance to glucose which starts or first becomes detectable during pregnancy. GDM is the most common metabolic illness, and impacts up to 25% of pregnant women. It has been demonstrated that omentin-1, which originates from both human placental and adipose tissue can serve as an insulin resistance mediator. It was initially identified as a secretory factor specific to visceral, both glucose and insulin can down-regulate omentin-1 which reduces levels in overweight women with polycystic ovarian syndrome. Moreover, those with diabetes and obesity had lower omentin-1 levels. Adipokines influence a variety of metabolic processes, including insulin sensitivity and secretion, inflammation, appetite control and adipogenesis regulation. This study was carried out to determine the serum omentin-1 level in pregnant women with and without gestational diabetes and determine any potential correlation between adipokine omentin-1 and gestational diabetes. In the case–control study, the cases collected (pregnant women in the third trimester of pregnancy) were divided into two groups **Group A**: 45 pregnant women with GDM as a patients. **Group B**: 45 apparent healthy pregnant women without GDM as control. Omentin-1 was determined by enzyme-linked immune sorbent assay ELISA. Serum levels of omentin-1 were significantly lower in patients with gestational diabetes mellitus compared with healthy pregnant women, $p < 0.05$, hence this biomarker can serve as a prognostic indicator for gestational diabetes. A lowered omentin-1 may play a role in the etiology of GDM. Its level as compared to healthy pregnant females was decreased in women with GDM, this could be due to reduced synthesis or increased elimination, but more research are needed to determine the exact mechanism.

Keywords: Gestational diabetes, Omentin-1, Pregnant women, Insulin resistance, Fasting blood glucose.

Introduction

Diabetes mellitus (DM) is the most prevalent metabolic disease characterized by a continuous increase in blood glucose levels and variable degrees of impaired protein, lipid, and carbohydrate metabolism [(1)]**.** Based on the International Diabetes Federation (IDF), there are 463 million individuals with diabetes globally as indicated in 2019, and this statistic is expected to increase to 700 million by 2045 [(2)]. Diabetes has been linked to long-term hyperglycemia, which can cause numerous problems, malfunctions, and even organ failure in the kidneys, heart, blood vessels, eyes, and nerves [(3)]. There are three different kinds of diabetes: Type 1 diabetes, Type 2 diabetes (T2DM), and Gestational Diabetes Mellitus (GDM) [(4)]. Glucose intolerance that starts or is first identified during pregnancy is known as gestational diabetic disease (GDM) [(5)]. In pregnancy, up to 25% of women may be influenced by GDM, the most prevalent metabolic condition [(6)]. GDM is a severe medical condition that develops during pregnancy [(7)]. And has many health complications for the mother as well as the child, such as premature birth, rapid fetal development, an elevated insulin level in newborns, hypoglycemia, and hyperbilirubinemia, among other conditions [(8)]. GDM is a kind of diabetes that typically develops within the second or third trimester of pregnancy [(9)]. The metabolic state undergoes major changes during pregnancy, which impacts the action and sensitivity of insulin. This effect is more pronounced in the second part of pregnancy because of hyperglycemia brought on by insulin resistance [(10)]. GDM progresses more quickly in women who are older than 25 years, have had GDM since their last pregnancy, and who also have a history of polycystic ovarian syndrome (PCOS) and T2DM [(11)]. There is still much to discover about the etiology of GDM, although it has been observed that certain ethnic groups of women, obesity, and older mothers are at greater risk for the disease [(6)]. To meet the fetus's energy needs, a woman's body goes through many physiological changes throughout pregnancy. In an attempt to improve the fetus's glucose supply insulin

Resistance increases. Pancreatic β-cells compensate for the increased demand for glucose, resulting in the development of a normoglycemic state. Conversely, women who have had GDM before are experiencing an inadequate β-cell response, which results in decreased insulin production and eventually, hyperglycemia. Thus, when β-cells lose their ability to control insulin resistance, glucose sensitivity may result [12]. The American Diabetes Association (ADA) suggests an oral glucose tolerance test (OGTT) for expectant mothers between 24 and 28 weeks of pregnancy, despite disagreements on the most effective GDM screening test [4]. By the guidelines provided forth by the International Association of Diabetes and Pregnancy Study Groups (IADPSG), routine screening and diagnosis of gestational diabetes mellitus can take place during week 24 and week 28 of pregnancy [13]. A 2-hour, 75-gram OGTT has been suggested by these guidelines as the standard GDM test [14]. Furthermore, based on large gestational age (LGA) concerns that refer to a fetus that is larger than expected for its age and gender, the IADPSG published updated diagnosis criteria for GDM in 2010. The concentrations of plasma glucose measured by 1-h, 2-h OGTT, and fasting blood glucose (FBG) were found to be 7.4, 6.2, and 4.5 mmol/l, respectively [15]. Gestational diabetes mellitus is a common pregnancy condition in which hyperglycemia develops randomly during pregnancy. Therefore, insufficient glucose tolerance brought on by pancreatic cell failure against the backdrop of persistent insulin resistance is the primary cause of hyperglycemia [16]. Adipokines are involved in numerous metabolic processes that are linked to inflammation, insulin sensitivity, appetite control, satiety control, and cardiovascular health [17]. Omentin-1, often referred to as intelectin-1(intestinal lactoferrin receptor, endothelial lectin HL-1, galactofuranose-binding lectin) is a glycoprotein that is one of several recognized adipokines. It has been identified as the primary secretory adipokine involved in visceral fat and has been suggested to influence lipid metabolism. It may play a role in regulating the breakdown of fats (lipolysis) and lipid storage in adipose tissue. By modulating lipid metabolism, omentin-1 could impact insulin sensitivity and overall metabolic health [17]. It exists as omentin-1 and omentin-2 isoforms. The predominant type seen in human blood is omentin-1. A chromosomal region (1q21.3) linked to type 2 diabetes contains the omentin-1 gene. This adipokine contributes to the emergence of inflammatory disease. It improves the functioning of the cardiovascular system, energy homeostasis, glucose metabolism, and oxidative stress reduction. Additionally, omentin-1 exhibits preventive properties against metabolic bone disease, atherosclerosis, and cancer [18]. Omendin-1 is a glycoprotein composed of N-linked oligosaccharides and 296 amino acids. Its major structural element is a 120 kDa homotrimer made up of 40 kDa polypeptides joined by disulfide bonds [19]. Omentin-1 may have a major paracrine or endocrine effect in regulating insulin sensitivity and activity because of its possible involvement in the metabolism of fats and carbohydrates [17]. This study aims to determine the serum omentin-1 level in pregnant women with and without gestational diabetes and investigate potential correlations between adipokine omentin-1 and gestational diabetes during the third trimester of pregnancy.

Materials and Methods

Patients

An observational case-control study was conducted on a cohort of Iraqi pregnant women with normal glycemic control and those with gestational diabetes mellitus GDM, who had been diagnosed with GDM during pregnancy. The participants in this study were chosen from the individuals seeking medical care at AL Alawiya Maternity Hospital in Baghdad, Iraq. The recruitment commenced in February 2023 and terminated in July 2023. The research protocol has been approved by the College of Pharmacy Scientific and Ethical Committee, University of Baghdad (RECAUBCP542023K), after the participant was given full information about the study's goal, they gave their informed consent. All the participants were interviewed by the researcher and demographic data were obtained from them and recorded in a data collecting sheet, including age, body weight and height, number of pregnancies, gestational age, and past medical history. A total number of 100 participants initially participated in the study. Nevertheless, the blood samples from ten patients were omitted from the study because of their hemolysis. The remaining ninety patients were categorized into two groups: **Group A** consisted of forty-five individuals who were diagnosed with GDM, serving as the patient group. **Group B** consisted of forty-five healthy pregnant women without GDM, serving as the control group.

Inclusion criteria

Patients were selected to be previously diagnosed with gestational diabetes mellitus according to International Association of Diabetes and Pregnancy Study Groups (IADPSG) diagnostic criteria [13, 15]. Diabetic pregnant patients must be above 18 years. HbA1c is $\geq$ (5.9%).

Exclusion criteria

- Women who suffer from other endocrinopathies rather than diabetes, women with cardiac, renal, liver, and autoimmune diseases. Women with malignancy or taking drugs that may interfere with glycemic control.
- Women with Inborn Errors of metabolism (glycogen storage disease, lysosomal storage disease).

Specimen collection and handling

Ten milliliters (ml) of venous blood were taken by venipuncture from each participant, who was a pregnant woman in the third trimester of pregnancy. Two milliliters of the blood sample were transferred into an ethylene diamine tetra acetic acid (EDTA) tube for the HbA1c assay. The remaining 8 mL of blood was transferred to a gel tube, centrifuged for 10 minutes at 3000 rpm, and allowed to coagulate for 30 minutes in to extract the serum. A portion of the serum was used by the hospital's laboratory to measure fasting blood glucose (FBG). Until the time that the levels of omentin-1 were measured, the remaining serum was frozen in Eppendorf tubes at -20°C.

Measurement of omentin-1 levels

Plasma omentin-1 levels were measured using commercial enzyme-linked immunosorbent assay (ELISA) kits (Elabscience, USA). Omentin-1 can be identified using this "sandwich enzyme immunoassay," which has a detection range of 0.63 to 40 ng/mL. The procedures have been carried out according to the manufacturer's instructions. Within and between assays, variations were less than 10%.

Other Biochemical Parameter

An enzymatic colorimetric approach was used to assess FBG [20]. *g*lycosylated hemoglobin (HbA1c) is biomarker of glycemic control in Diabetes Mellitus (DM) condition because it describes blood glucose levels in the last 60-90 days [21]. Fasting serum insulin (FSI) was measured by using Commercial competitive inhibition enzyme-linked immunosorbent assay (ELISA) kits (Cloud-Clone-Corp). The results of FSI and FBG concentrations were used to calculate an index of insulin resistance: HOMA-IR (Homeostasis Model Assessment—Insulin Resistance):

$$HOMA-IR = glycemic\ (mmol/L) \times fasting\ insulin\ (mU/L)/22.5$$ [18].

Statistical Analysis

Statistical analysis was performed using the Statistical Package for the Social Science (SPSS, IBM, USA, version (25).The Shapiro–Wilk test was used to test the normality of the results. Continuous variables were expressed as mean ± SD of the values. For normally distributed groups, the mean differences of two independent groups were measured by unpaired t-test. Pearson correlation was performed to determine the relation between omentin-1 and study variables, and the correlation coefficient (*r*) was used to calculate the association between parameters. A probability that equals or less than (0.05) indicates a significant difference.

Results

The demographics information compares two groups of pregnant women: one with gestational diabetes (GDM) and the other healthy without gestational diabetes (NGDM).

By the measurement of the *p*-value for the mean values of GDM and NGDM, among study groups were demonstrated statistically significant differences for age, Body Mass Index (BMI), No. of pregnancy, and gestational age between these groups as presented in "Table 1".

Table 1. Demographic data for pregnant women with no gestational diabetes (NGDM) and pregnant women with gestational diabetes (GDM).

Characteristics	NGDM (N=45)	GDM (N=45)	*P*-value
Age (Years) Mean ± SD	27.76±4.86	31.56±6.91	0.003*
BMI (kg/m^2) Mean ± SD	23.28±2.56	26.30±2.47	0.0001*
Gestational age (weeks) Mean ± SD	35.40±3.87	33.36±4.35	0.021*
No. of pregnancy Mean ± SD	3.42±1.56	4.78±2.34	0.002*

NGDM: pregnant women with no gestational diabetes, GDM: pregnant women with gestational diabetes, N: number, BMI: body mass index, SD: standard deviation, No. of pregnancy: number of pregnancy *: significance difference (P<0.05)

Biochemical characteristics of pregnant women with and without gestational diabetes

The result of FBS data showed significantly higher levels in pregnant women with GDM and those without GDM HBA1c, fasting serum insulin, and HOMA-IR also showed significantly higher levels in pregnant women with GDM and those without GDM as presented in "Table 2".

Table 2. Biochemical characteristics for pregnant women with no gestational diabetes (NGDM) and pregnant women with gestational diabetes (GDM)

Marker	NGDM (N=45) Mean ± SD	GDM (N=45) Mean ± SD	*P*-value
FSI mU/L	3.24±0.67	7.76±3.52	0.0001***
FBG mmol/L	4.74±0.63	8.12±3.10	0.0001***
HbA1c%	4.76±0.35	6.91±1.19	0.0001***
HOMA-IR	0.68±0.15	2.82±1.68	0.0001***
Omentin-1 (ng/ml)	26.70±5.08	12.05±1.58	0.0001***

NGDM: pregnant women with no gestational diabetes, GDM: pregnant women with gestational diabetes, FBG: Fasting blood glucose, FSI: Fasting serum insulin, HOMA-IR: Homeostasis model assessment of insulin resistance, N: number, SD: standard deviation ng: nanogram , ml: milliliter, mmol: milli mole, L: Liter, *P* value < 0.05 is significant, **: highly significant difference (*P*<0.01). ***: very highly significant difference (*P*<0.001).

Serum Levels of omentin-1 in diabetic pregnant women

A statistically significant difference was seen in the serum omentin-1 levels between the two participating groups ($p<0.05$, unpaired t-test). Omentin-1 serum levels were significantly lower in GDM patients (mean= 12.05± 1.58 ng/mL) than in the non-GDM groups (26.70 ± 5.08 ng/mL; $p<0.05$). These results indicated a relationship between lower serum omentin-1 levels and the development or progression of GDM. Additionally, a correlation has been observed between blood levels of omentin-1 and the severity of GDM.

Correlations between parameters and biomarkers included in the current study

The results illustrated in "Table 3" show Pearson's correlation analysis for the association between the serum biomarker levels. A significant negative association was found among serum levels of omentin-1 and studied variables.

Table 3. Spearman's correlation of serum omentin-1 with the studied variables for pregnant women with and with no gestational diabetes mellitus

Variables	r- value	*p*- value
FBG	-0.557****	0.000
HbA1c	-0.712****	0.000
FSI	-0.565****	0.000
HOMA-IR	-0.582****	0.000

FBG: fasting blood glucose, HbA1c: glycated hemoglobin, FSI: fasting, serum insulin, HOMA-IR, * significant when the p-value of spearman correlations was <0.05, ***: very highly significant difference *(P*<0.01)

Discussion

According to the current study, women with GDM had lower serum Omentin-1 levels than women who were pregnant healthily. This study supports a different one that found that women with gestational diabetes mellitus have lower serum adiponectin concentrations than pregnant women in good health. The study's subjects were Iraqi pregnant women from the National Diabetes Center for Treatment and Research at Al-Mustansiriya University and Al-Yarmuk Hospital in Baghdad [22]. The current study's results also supported earlier research showing that GDM patients had lower levels of circulating omentin-1 than those controls. Reduced levels of omentin-1-concentration, which are secreted by visceral adipose tissue, may lead to IR and GDM [23]. Omentin-1, which is produced from human placental and adipose tissue, has been suggested as a possible mediator of insulin resistance. Yang et al. initially characterized it as a secretory factor that was particular to visceral fat. They specifically showed that recombinant omentin-1 administration increased insulin-stimulated glucose transport in vitro, indicating that omentin-1 may enhance insulin sensitivity [24]. Considering of the placenta also produces omentin-1 appears unexpected. Further investigation is necessary to determine if the decline in omentin-1 levels throughout pregnancy is due to normal hemodilution during gestation or an enhanced clearance in the latter stages of pregnancy. Conversely, it has been demonstrated that insulin and glucose down-regulate omentin-1. Thus, it stands to reason that both women with GDM and those without it would experience lower levels of omentin-1 in the third trimester as a result of their altered insulin sensitivity during pregnancy [24]. Finding from the study, reports that fasting blood sugar, fasting serum insulin, and insulin resistance were significantly higher in GDM patients than in women with healthy pregnancies, which agreed with the previous studies: [25, 26]. Elevated insulin resistance may rise from decreased insulin sensitivity, which is typically observed during

pregnancy to protect the fetus's glucose reserves. This is therefore linked to the impact of hormones secreted by the placenta, as well as physiological changes in some pregnant women that result in impairment to their glucose tolerance, which may cause gestational diabetes mellitus [27]. Physiological insulin resistance can be shown with increasing gestational age due to the placenta's production of lactose, estrogens, progesterone, and maternal adrenocorticotropic hormones [26]. However, other research suggested that the primary cause of insulin resistance in GDM is post-cellular damage, which is shown by decreased tyrosine phosphorylation in insulin receptors and insulin receptor substrate-1, while increased serine phosphorylation inhibits insulin signaling by preventing glucose transporter type 4 (GLUT4)-translocation [28]. Additionally, compared to pregnant women without GDM, pregnant women with GDM may have worse insulin sensitivity by 30–40% and increased peripheral insulin resistance, primarily in skeletal muscle, as well as reduced insulin secretion. Additionally, the secretion of insulin is significantly reduced in response to hyperglycemia, suggesting a primary beta cell deficiency that complicates the process of compensating for elevated insulin resistance and suggests multiple insulin action deficiencies in the long term. These factors contribute to the etiology of gestational diabetes [28]. The whole studied population (n=90) had a correlation analysis conducted to determine how serum omentin-1 concentration related to FBG, FSI, HbA1c, and HOMA-IR. The results indicated a significant negative correlation between omentin-1 concentration and the parameters mentioned above, as "Table 3" illustrates. As serum concentrations of FBG, FSI, HbA1c, and HOMA-IR increased, the serum omentin-1 concentration decreased. Although the exact mechanism underlying this process is yet unknown, it is believed that lower omentin-1 concentrations may facilitate the emergence of insulin resistance [18]. Some limitations in the study including: Initially, this study was carried out at a single center so additional long-term clinical studies are required to assess whether omentin-1 level detection aids in early diagnosis and prognosis, and whether lower serum levels of Omentin-1 increases the risk of GDM in pregnant women. Secondly, lifestyle factors like exercise and diet were not considered. Third, difficulties with getting pregnant women to provide information and a blood sample.

Conclusion

The case-control study findings support omentin-1 as a biomarker for the early detection of GDM, a condition that affects a large number of pregnant women. A lack of omentin-1 may play a role in the etiology of GDM. When compared to healthy controls, women with GDM had lower levels of omentin-1. This could be due to reduced synthesis or increased release, but more research is needed to determine the exact mechanism. Omentin-1's precise methods of action in glucose metabolism remain unclear as of the moment. Furthermore, Omentin-1 was negatively correlated with HbA1c, FSI, HOMA-IR, and FBG. To overcome the challenges, we hope to conduct a large-scale prospective study in the future.

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Conflicts of Interest

The authors declare that there is no conflict of interest

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Ethics Statements

This study was approved by the Ethical and Scientific Committee of the College of Pharmacy at the University of Baghdad, Iraq with Ethical approval with the (RECAUBCP542023K).

Author Contribution

All authors reviewed the results and approved the final version of the manuscript.

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تقييم مستوى الاومنتين-١ في مصل الدم لدى النساء العراقيات المصابات بسكر الحمل

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[١] فرع العلوم المختبرية السريرية ،كلية الصيدلة ،جامعة بغداد، بغداد، العراق

الخلاصة

تصف الحالة المعروفة باسم داء السكري الحملي بأنها عدم تحمل الجلوكوز الذي يبدأ أو يصبح قابلاً للاكتشاف لأول مرة أثناء الحمل. داء السكري الحملي هو مرض التمثيل الغذائي الأكثر شيوعًا، ويؤثر على ما يصل إلى ٢٥٪ من النساء الحوامل. لقد ثبت أن الأومينتين-١، الذي ينشأ من كل من الأنسجة المشيمية والدهنية البشرية، يمكن أن يكون بمثابة وسيط لمقاومة الأنسولين. تم تحديده في البداية على أنه عامل إفرازي خاص بالحشوية، حيث يمكن لكل من الجلوكوز والأنسولين تنظيم الأومينتين -١ مما يقلل مستوياته لدى النساء ذوات الوزن الزائد المصابات بمتلازمة المبيض المتعدد الكيسات. علاوة على ذلك، كان لدى المصابين بالسكري والسمنة مستويات أقل من الأومينتين-١. تؤثر الأديبوكينات على مجموعة متنوعة من العمليات الأيضية، بما في ذلك حساسية الأنسولين وإفرازه، والالتهابات، والتحكم في الشهية وتنظيم تكوين الدهون. أجريت هذه الدراسة لتحديد مستوى أومينتين-١ في الدم لدى النساء الحوامل المصابات وغير المصابات بسكري الحمل وتحديد أي علاقة محتملة بين أديبوكين أومينتين-١ وسكري الحمل. في دراسة السيطرة على الحالة، سيتم تقسيم الحالات التي تم جمعها (النساء الحوامل في الأشهر الثلاثة الأخيرة من الحمل) إلى مجموعتين المجموعة أ: ٤٥ امرأة حامل مصابة بداء السكر الحملي كمريضة. المجموعة ب: ٤٥ امرأة حامل تتمتع بصحة جيدة ولا تعاني من مرض السكري كمجموعة سيطرة. يتم تحديد الأومينتين -١ بواسطة مقايسة الامتصاص المناعي المرتبط بالإنزيم. كانت مستويات الأومينتين-١ في مصل الدم أقل بشكل ملحوظ في المرضى الذين يعانون من داء السكري الحملي مقارنة بالمجموعة الضابطة، (قيمة الاحتمالية <٠,٠٥) وبالتالي يمكن أن يكون هذا المؤشر الحيوي بمثابة مؤشر إنذار لمرض السكري الحملي. قد يلعب نقص أومينتين-١ دورًا في مسببات مرض داء السكر الحملي. عند مقارنتها بالضوابط الصحية، كان لدى النساء المصابات بداء السكر الحملي مستويات أقل من أومينتين-١، وقد يكون هذا بسبب انخفاض التوليف أو زيادة الإطلاق، ولكن هناك حاجة إلى مزيد من البحث لتحديد الآلية الدقيقة.

الكلمات المفتاحية: سكري الحمل، أومينتين-١، النساء الحوامل، مقاومة الأنسولين، جلوكوز الدم الصائم