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Adiponectin , β-Cell Dysfunction in Iraqi Women with Gestational Diabetes

Fayhaa M. Khaleel* ***Isam N. Salman**** ***Hawraa I. Kadhim****

*Department of Chemistry, College of Science for Women, University of Baghdad, Baghdad, Iraq.
** National Diabetes Center (NDC), Al-Mustansiria University, Baghdad, Iraq

E-mails : fyaha_magdad@yahoo.com

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Abstract:

Gestational diabetes mellitus (GDM) is a complication of gestation that is characterized by impaired glucose tolerance with first recognition during gestation. It develops when β- cell of pancreas fail to compensate the diminished insulin sensitivity during gestation. This study aims to investigate the relationship between mother adiponectin level and β- cell dysfunction with development gestational diabetes mellitus (GDM) and other parameters in the last trimester of pregnancy. This study includes (80) subjects (pregnant women) in the third trimester of pregnancy, (40) healthy pregnant individuals as control group aged between (17 - 42) years and (40) gestational diabetes mellitus patients with aged between (20 - 42) years. The following biochemical investigation is studied: oral glucose tolerance test (OGTT), adiponectin , insulin, C-reactive protein (CRP),body mass index (BMI), and homeostasis model assessment- insulin resistance (HOMA – IR). The adiponectin levels are significantly lesser in females who develop GDM than the control group ($P \leq 0.01$), while the insulin and OGTT concentrations were significantly higher in females with GDM than control group ($P \leq 0.01$).The concentrations of CRP are non significantly different between the females who develop GDM and the control group. Conclusions: Lower adiponectin concentrations are associated with an increased risk of the development of gestational diabetes mellitus and females, who develop gestational diabetes mellitus, have higher levels of insulin resistance from normal females, Obesity is a shape of persistent low grade inflammation which causes elevated concentrations of C- reactive protein.

Key words: Gestational Diabetes Mellitus (GDM), Adiponectin, Beta Cell Dysfunction, Insulin, Oral Glucose Tolerance Test (OGTT), C-Reactive Protein (CRP).

Introduction:

Gestational diabetes mellitus (GDM), distinguished as glucose intolerance with onset or first identification during gestation, is a widespread complication

of gestation . Females with a history of gestational diabetes mellitus have a seven fold raised risk of developing type two diabetes mellitus (T2DM) after parturition , and the babies of females with GDM are more likely to be overweight and develop diabetes mellitus [1, 2]. The risk of development T2DM in females with a history of GDM may be connected with raised insulin resistance and subclinical inflammation [3]. Gestational diabetes mellitus and type 2 diabetes mellitus have the same pathophysiology, the two major metabolic fault underlying both GDM and T2DM are target cell resistance to the effect of insulin and inadequate secretion of insulin by the pancreatic β-cells. Like that untreated patients are incapable to maintain blood glucose levels in normal value [4].For keeping glucose homeostasis in normal state, the pancreatic β-cells requires to increase insulin secretion to compensate insulin resistance [4]. In pregnancy state, the β-cells must raise their insulin secretion to match (50%-60%) ,as a result this reduces insulin sensitivity that occurs in the last trimester of pregnancy . Females with GDM have an underlying persistent β-cell fault like their compensatory raise in insulin secretion is inadequate to offset the insulin resistance of last gestation [5]. Adiponectin is a protein hormone produced by white adipose tissue and shown to be down-regulated in conditons of insulin resistance [6]. It is anti-atherosclerosis, anti-diabetes mellitus , anti-tumor effective, and anti-inflammation linked directly to the high molecular weight adiponectin [7].Moreover, it is linked by the receptors AdipoR1 and AdipoR2 [8] , AdipoR1 is expressed in muscle, while AdipoR2 is abundantly expressed in the liver [9] . Adiponectin modifies multi metabolic processes, out of these are glucose regulation and fatty acid oxidation . This hormone is exclusively secreted from adipose tissue (and in pregnancy secreted also from the placenta [10]) into the bloodstream and is very abundant in blood relative to numerous hormones. Concentrations of the adiponectin are inversely correlated with body fat proportion in adults [11] , while circulating hormone levels raises during caloric restriction, such as in patients with anorexia nervosa. This observation ,unexpectedly is given that adiponectin is created by adipose tissue; though, a new study proposes that adipose tissue within bone marrow, which elevates in the state of caloric restriction, contributes to increase circulating adiponectin in this condition [12]. Adiponectin plays a role in the inhibition of the metabolic disruption that may result in T2DM [11] , atherosclerosis, obesity , an independent risk factor for metabolic syndrome and non-alcoholic fatty liver disease. Adiponectin, abundantly present in the plasma, raises insulin sensitivity by stimulating fatty acid oxidation, diminishes plasma triglycerides and inhances glucose metabolism [13]. C-reactive protein is an ordinary pentameric protein found in blood, the levels of C-reactive protein (CRP) in circulating blood increase noticeably a cytokine- mediated in the

most states of tissue damage, inflammation and disease. CRP is produced by the liver in response to factors released by macrophages and adipocytes and can measure the level of CRP in clinical practice as a point index of illness activity [14]. The present study aims to investigate the relationship between mother adiponectin level and β- cell dysfunction with development gestational diabetes mellitus (GDM) and other parameters in the last trimester of pregnancy.

Materials and Methods:

Subjects :

For this study, 40 females with GDM are selected and 40 well matched females without GDM as the control group.The subjects are from the National Diabetes Center for Treatment and Research at Al-Mustansiriya University and Al- Yarmuk Hospital located in the city of Baghadad, Iraq from October 2014- March 2015. Women with GDM whose treatment by insulin injection and the pregnant women in the first trimester are excluded from the study. Diagnosis of gestational diabetes mellitus is made on the basis of the recommended criteria by WHO [15].

Specimens, Collection, and Evaluation

Blood collected from each subject at (8 AM – 1 PM), 3ml of vein puncture blood sample is taken in fasting state , 2ml after 1 hour from drinking glucose solution , 2ml after 2 hours and 2ml after 3 hours using in each time 5ml disposable syringe . The first blood sample (in the fasting state) is divided into two aliquots; 1 and 2 ml. The first aliquot is dispensed in tube containing ethylene diamine tetracetic acid (EDTA). This blood is processed in less than 3 hours and used for plasma glucose estimation after centrifugation. The second aliquot is dispensed in a plain tube and left for around an hour to clot at room temperature. Then, it is centrifuged at 3000 rpm for 10 minutes to collect serum. The serum is divided into aliquots in Eppendorff tubes and stored in the freezer (-20ºC) until being used for estimation of Adiponectin , insulin and C-reactive protein. The collected blood after 1 hour, 2 hour and 3 hour from drinking glucose solution is dispensed in tube containing ethylene diamine tetracetic acid (EDTA) and this blood is used for plasma glucose estimation at the same day.

Statistical Analysis

between difference parameters in this study is also done .Cary. N.C.(2012). Statistical Analysis System, User's Guide. Statistical version 91th ed. SAS. Inst. Inc.. USA. The Statistical Analysis System- SAS (2012) uses different factors in studying the parameters. Least significant difference –LSD test is used to significantly compare between means. Estimate of correlation coefficient.

Results

The present study has found out a significant differences in the mean of serum adiponectin , insulin and plasma glucose levels in OGTT between females with GDM and the control groups (Table 2). Beside, there is no significant difference in the mean of serum CRP between females with GDM and the control group (Table 2).

Table (1): Demographic and Characteristics of Patients and Control Groups.

Parameters	Patients group (n=40)	Control group (n=40)
Age in (year)	28.91 ± 3.72	27.73 ± 4.02
BMI Kg/m^2	33.44 ± 3.74	31.97 ± 3.28
HOMA – IR	5.37 ± 0.11	2.094 ± 0.06

Values as mean ± SE

Table (2): The Mean ± SE of Serum Adiponectin, Insulin, C-Reactive Protein and Plasma OGTT Levels for GDM Patients and Control Groups.

Parameters	Patients group (n=40)	Control group (n=40)	P value
Adiponectin (µg/l)	67.71 ± 4.80	85.93 ± 4.94	0.01
Insulin (µU/l)	17.88 ± 1.21	9.77 ± 1.06	0.01
CRP(mg/l)	9.23 ± 0.94	8.71 ± 0.94	NS
OGTT: fasting (mg/ dl)	121.13 ± 3.32	84.70 ± 1.42	0.01
OGTT1: 1 hr. (mg/dl)	205.67 ± 5.17	125.35 ± 3.31	0.01
OGTT : 2 hr. (mg/dl)	175.75 ± 5.06	103.77 ± 2.49	0.01
OGTT: 3 hr. (mg/dl)	126.05 ± 4.90	83.85 ± 1.69	0.01

Significant at $p<0.01$, non significant (NS)

Table (3): Correlation Coefficients Between Adiponectin and Parameters of Study.

Parameters correlated with Adiponectin	Correlation coefficients (r)	Level of sig.
CRP (mg/l)	0.02	NS
Insulin (µU/l)	-0.26	**
OGTT: fasting (mg/dl)	-0.23	*
OGTT: 1 hr. (mg/dl)	-0.27	**
OGTT : 2 hr. (mg/dl)	-0.22	*
OGTT :3 hr. (mg/dl)	-0.21	*
Age (year)	-0.13	NS
HOMA-IR	-0.06	NS
Weeks of gestation	0.01	NS
BMI	-0.04	NS
Parity	-0.17	NS
Weight baby	-0.01	NS

* (P<0.05), ** (P<0.01), NS: Non-significant.

Discussion

The present study shows that serum adiponectin concentrations diminish in females with gestational diabetes mellitus more than healthy pregnant women. This agrees with another study which demonstrats that both high molecular weight (HMW) adiponectin and total adiponectin levels are significantly lower in females who develop gestational diabetes mellitus in comparison with women who do not develop [1]. Low concentrations of adiponectin are related with lipid abnormalities, inflammation, insulin resistance , raised risk of diabetes mellitus and risk of several types of cancer [16]. Some studies suggest that as body mass index raises, blood adiponectin concentrations decline [17] .The larger adipocytes found in obese individuals, particularly those with visceral obesity ,create lower concentrations of adiponectin [18], but higher concentrations of some cytokines, such as tumor necrosis factor alpha (TNFα) [19, 20]. Adipocyte expression and secretion of adiponectin are shown to be decreased by tumor necrosis factor alpha . In the current study, both patients and control females are obese and the result shows that there is a significant difference ($p\leq 0.01$) between patients and control pregnant women in the mean of adiponectin levels independent of the body mass index (BMI). This agrees with diverse studies which demonstrat that circulating adiponectin concentrations are decreased in pregnant females with gestational diabetes mellitus in comparison with healthy pregnant women (without gestational diabetes) independent of body mass index before gestation [21 ,22]. In agreement with these conclusions, adiponectin messenger RNA is down regulated in placental tissue in females with gestational diabetes mellitus [23]. Furthermore, down regulation of

adiponectin in the first three months of gestation is an independent predictor of impending gestational diabetes mellitus. Thus, Lain et al. [24] suggest that females with first three months adiponectin levels under the 25th percentile are ten times more likely to be diagnosed with gestational diabetes mellitus as compared to females with elevated adiponectin concentrations. Gestational diabetes mellitus commonly diagnosed in the second or third trimester of gestation. Insulin necessities are raised during gestation because of the existence of insulin antagonists, such as lactogen secreted from human placenta, these hormones activate lipolysis and diminish glucose use. Diverse genetic disorder of the β- cells, insulin function, diseases of the exocrine pancreas, endocrinopathies, can cause changeable degrees of glucose intolerance .So, the glucose intolerance increases in GDM when β- cells are unable to compensate the elevated insulin resistance in gestation . In T2DM the cells of the body become resistant to the action of insulin as the receptors that bind to the hormone become less sensitive to insulin levels.This cause an increase in insulin levels (hyperinsulinemia) and disorders in releasing of insulin , with a lessened response to insulin. The beta cells release raised concentrations of insulin in response to the continued elevated blood glucose concentrations resulting hyperinsulinemia in blood. The hallmark of gestational diabetes mellitus raise insulin resistance. Gestational hormones and additional factors are thought to interfere with the effect of insulin, as it binds to the insulin receptor. This interfering may occur at the level of the cell signaling pathway following the insulin receptor [25]. Accurate measures of insulin sensitivity applied in the last trimester show slightly larger insulin resistance in females with gestational diabetes mellitus than in healthy pregnant females. The further resistance occurs for insulin's effects to stimulate glucose elimination and to suppress glucose creation and decrease fatty acid concentrations [26] . So the levels of insulin in pregnant females with gestational diabetes mellitus are larger than insulin levels in control pregnant females due to the increased levels of insulin resistance in women with gestational diabetes mellitus. Another study establishes that mother blood concentrations of C- reactive protein are not correlated to gestational diabetes at the time of OGTT in late second or early last trimester. The concentration of C- reactive protein relates strongly with pre-pregnancy mother obesity, consistent with new notes in normoglycemic pregnant females [27]. Many studies have firmly confirmed the strong association between high levels of inflammatory markers and obesity, leading to the recognition of obesity as a shape of persistent low grade inflammation [28, 29]. Circulating concentrations of C-reactive protein relates strongly with many measures of body fat, including body mass index , fat free mass, adipose body mass and waist circumference [30]. Mother obesity has been connected with the up-regulation of inflammatory markers such as C-reactive protein and interleukin six in the first three months from gestation prior to any observed glucose dysregulation [31]. Tumor necrosis factor alpha increased in obesity induces expression of interleukin 6, and the principal determinant of hepatic C-reactive protein expression . Many studies have demonstrated high concentrations of interleukin 6 and C-reactive protein among subjects both with features of the insulin resistance and clinically overt type two diabetes mellitus [32].The recent study confirms that C-reactive protein is a precise predictor for insulin resistance in diabetic individuals [33].It also shows

increased CRP levels in obese females independently with GDM.

Conclusions:

Lower adiponectin concentrations are associated with an increased risk of development of gestational diabetes mellitus and females who develop gestational diabetes mellitus have higher levels of insulin resistance from normal female. Obesity is a shape of persistent low grade inflammation which causes elevated concentrations of C- reactive protein**.**

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الاديبونكتين ،الاختلال الوظيفي لخلايا بيتا عند النساء العراقيات المصابات بسكري الحمل

فيحاء مقداد خليل* **عصام نوري سلمان****
حوراء عماد كاظم*

*قسم الكيمياء، كلية العلوم للبنات، جامعة بغداد، بغداد، العراق
** المركز الوطني للسكري ،الجامعة المستنصرية،بغداد، العراق

الخلاصة:

سكري الحمل هو من اضطرابات الحمل التي تتصف بضعف تحمل الكلوكوز لحظة تشخيصه خلال الحمل . و يتطور عندما تفشل خلايا بيتا في مواكبة زيادة طلب الأنسولين نتيجة النقص الحاصل في حساسية الأنسولين خلال فترة الحمل. هذه الدراسة تهدف إلى التحقق من العلاقة بين مستوى هرمون الأديبونكتين في دم الأم و الخلل الوظيفي لخلايا بيتا مع تطور سكري الحمل و متغيرات أخرى في خلال الأشهر الثلاث الأخيرة من الحمل. هذه الدراسة شملت (80) عينة (نساء حوامل) في الأشهر الثلاث الأخيرة من الحمل. (40) عينة من النساء الحوامل اللواتي بصحة جيدة كمجموعة سيطرة والتي يتراوح معدل أعمارهن بين (17- 42)، و (40) من النساء المصابات بسكري الحمل مع معدل أعمار يتراوح بين (20-42). المتغيرات الحيوية التالية هي التي تم قياس مستواها في هذه الدراسة : اختبار تحمل الكلوكوز في البلازما و مستوى الاديبونكتين و الأنسولين و البروتين المتفاعل -C في مصل الدم . و كذلك معدل كتلة الجسم (BMI) و تقييم نموذج التوازن- مقاومة الأنسولين (HOMA-IR).مستوى الأديبونكتين في النساء المصابات بسكري الحمل أقل من النساء الغير مصابات بالمرض بفرق معنوي كبير ($P\leq0.01$) بينما مستوى الأنسولين و اختبار تحمل الكلوكوز أعلى معنوياً ($P\leq0.01$) في النساء المصابات بسكري الحمل من النساء الغير مصابات بالمرض. ليس هنالك فرق معنوي في معدل مستوى البروتين المتفاعل- C بين النساء المصابات بسكري الحمل و النساء الغير مصابات بالمرض.

الاستنتاج:

انخفاض مستوى الاديبونكتين في الدم يرتبط مع زيادة خطر تطور الإصابة بسكري الحمل و أن مستوى مقاومة الأنسولين في النساء المصابات بسكري الحمل أعلى من مستواه في النساء الغير مصابات بالمرض، السمنة هي نوع من الالتهاب المستمر بدرجة واطئة حيث تسبب ارتفاع في مستويات البروتين المتفاعل- C في الدم.

الكلمات المفتاحية: سكر الحمل، الأديبونكتين، فشل خلايا بيتا، الأنسولين، اختبار تحمل الكلوكوز، البروتين المتفاعلC.