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## The Link between Serum Omentin Level and Insulin Resistance Biomarkers, Lipid Profile, and Atherogenic Indices in Iraqi Obese Patients.

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### Abstract:

Omentin (or intelectin) is a main visceral fat secretory adipokine. There is a growing interest to link omentin, obesity and co-morbidity factors. The aim of the present study is to evaluate serum omentin and its association to insulin resistance biomarkers, lipid profile and atherogenic indices. This cross – sectional study was conducted in Obesity Research and Therapy Unit-Alkindy College of Medicine by recruiting (115) individuals; 49 males /66 females. Subjects between (20 to 60) years of age were selected and classified into two groups according to their Body mass index (BMI). Group1 involved healthy lean volunteers (25 male/ 36 female; BMI 18.5 - 24.9). Group2 involved obese subjects; (24 male / 36 female with BMI  $\geq$  30). The study shows that obese group has higher omentin levels compared to the healthy lean group ( $15.49 \pm 4.20$  vs.  $10.15 \pm 5.04$  pg/ml,  $P < 0.001$ ). In addition; obese group exhibited significantly–higher levels waist circumference (WC), waist to hip ratio (WHR), glucose, insulin, homeostatic module of insulin resistance (HOMA2-IR) and lipid profile and lower levels of HDL-Chol ( $P < 0.05$ ). Omentin levels were significantly and positively correlated with WC, WHR, BMI, glucose, hemoglobin A1c, HOMA2-IR, triglyceride, total cholesterol, low density lipoproteins - cholesterol, triglyceride to glucose index (TyG index) and atherogenic index of plasma (AIP); ( $P < 0.05$ ). Multiple regression analysis established that omentin levels was found to be associated with glucose, total Chol, TyG index and AIP in total population. These findings indicate that serum omentin levels are higher in obese individuals compared to lean subjects. Furthermore, omentin was linked to insulin resistance biomarkers and other risk factors indices such as AIP and TyG. Omentin can be used as a metabolic marker in obese.

**Keywords:** Atherogenic Index of Plasma, Insulin resistance, Lipid profile, Obesity, Omentin, Triglyceride to Glucose index.

### Introduction:

Obesity and visceral fat are healthy burden and major risk factors for the development of cardiovascular diseases, diabetes and other health complications<sup>1,2</sup>. Adipose tissue is important to medical research for its role as secretory organ of several proteins or cytokines that are important in cell signaling which identified as adipocytokines or adipokines<sup>3</sup>.

Adipokines participate in many metabolic processes related to insulin sensitivity, appetite regulation, satiety control, cardiovascular function and inflammatory processes. Clinically circulatory

levels of adipokines are regarded as markers of adipose tissue function and indicators of an increased metabolic defects<sup>4</sup>. Dysfunction of adipose tissue is an important prime defect seen in obese subjects and this reflect changes in circulatory levels of adipokines<sup>5</sup>.

One of these identified adipokines is a protein known as omentin or called intelectin. It has been recognized as a main visceral fat secretory adipokine<sup>6</sup>. Omentin is coded by two main genes (1 and 2). Omentin1or / intelectin1 is the circulating

form of this adipokine. Yet, its metabolic role still under evaluation <sup>7</sup>.

Omentin has a potential role in carbohydrate and lipid metabolism, consequently, omentin may show a significant paracrine or endocrine action in modulating insulin sensitivity and action <sup>1</sup>. There is a growing interest to link adipokines, obesity and co-morbidity factors. In addition, there are increasing needs to identify adipokines roles as biomarkers for evaluation and treatment of obesity. Accordingly, the aim of the present study is to evaluate serum omentin level (omentin 1) in a group of obese patients and compare them to healthy lean subjects and to elucidate the relationship between omentin and insulin resistance biomarkers, lipid profile; and atherogenic indices.

### Materials and Methods:

This cross – sectional study was conducted in Obesity Research and Therapy Unit (ORTU) – Alkindy College of Medicine – University of Baghdad from September 2019 to January 2020 by recruiting (115) individuals. The study protocol and ethical approval were permitted by Alkindy College of Medicine. Informed consent was obtained from all subjects involved in the study.

Subjects between (20 to 60) years of age were selected and classified into two main groups according to their Body mass index (BMI - calculated as weight in kilograms divided by height in squared meters). Group1 involved healthy lean volunteers (BMI 18.5 - 24.9); group2 involved obese subjects (BMI  $\geq$  30). Subjects who had malignancy, diabetes, or major renal disease, hepatic, or endocrine and thyroid dysfunction were excluded. Blood samples were drawn in the fasting state (for at least 12 hours) and centrifuged. Then serum samples were collected to be stored at -20°C until analytical processing.

Patients' health status data were acquired from medical records and augmented with self-reported information from the research participants. Blood pressure and anthropometric measurements; body weight (Kg) and height (m); circumference of waist and hip (cm). In addition, BMI (kg/m<sup>2</sup>) and waist to hip ratio (WHR) were estimated according to medical standards in the ORTU.

### Assessment of Biochemical Parameters:

Serum omentin (Human omentin) concentrations were measured by a commercially-available enzyme-linked immunosorbent assay (ELISA), using the kit provided by the manufacturer (CUSABIO TECH. CO. LTD., China). Serum glucose (S.Glucose), cholesterol (Chol),

triglycerides (TG), and high-density lipoproteins cholesterol (HDL-Chol) were measured using diagnostic kits produced by (Human, Germany). Serum insulin levels were estimated by ELISA kits (Demeditec Diagnostics., Germany). Glycated hemoglobin A1c was assessed by kits and HumaMeterA1c system analyzer manufactured by (Human, Germany). Each biochemical parameter was estimated according to the instructions provided with kits.

The following parameters were calculated using specific equations or online calculators:

1. Low-density lipoprotein cholesterol ((LDL-Chol) was calculated by the Friedewald Eq .1<sup>8</sup>:  
$$\text{LDL- cholesterol (mg/dL)} = \text{total cholesterol} - \text{HDL cholesterol} - (\text{triglycerides}/5) \dots \text{Eq .1}$$
2. The Triglyceride to glucose index (TyG index) was calculated by Eq. 2 <sup>9</sup>:  
$$\text{TyG index} = \text{Ln} [\text{Fasting TG (mg/ml)} * \text{Fasting Glucose (mg/dl)} / 2] \dots \text{Eq .2}$$
3. Non-HDL-Chol was calculated using the Eq .3 <sup>10</sup>:  
$$\text{Non-HDL-Chol} = \text{total cholesterol} - \text{HDL cholesterol} \dots \text{Eq .3:}$$
4. Atherogenic Index of Plasma was calculated by Eq .4:  
$$(\text{AIP} = \text{Log (TG/HDL-Chol)}) \dots \text{Eq .4}$$

The online calculator was used to estimate values of atherogenic index <sup>11</sup>.

### Estimation of Insulin Resistance Biomarker:

Fasting s. glucose and s. insulin levels were used to estimate the Homeostatic model assessment of insulin resistance (HOMA2-IR) <sup>12</sup>.

### Statistical analyses:

Data were analyzed with SPSS version 25 software for windows 10 (IBM, New York, NY, USA). Parametric data are presented as mean  $\pm$  Standard Deviation (mean $\pm$ SD). Independent Student's *t*-test was used to compare variables between groups, respectively. A (*P*) value <0.05 was used as the level of significance. Pearson's correlations were used to determine the relationship between serum omentin levels and the other variables. Linear regression analysis was undertaken to determine independent associations between Omentin and other variables included in the study.

**Results:**

The clinical characteristics and biochemical analytes of participants in the present study are shown in Table.1. The obese group has higher omentin levels when compared to the healthy lean group ( $15.49 \pm 4.20$  vs.  $10.15 \pm 5.04$  pg/ml,  $P$

$<0.001$ ). Moreover, the obese group exhibited significant higher levels of blood pressure, waist circumference (WC), waist to hip ratio (WHR), BMI, glucose, insulin, HOMA2-IR and lipid profile and lower levels of HDL-Chol ( $P < 0.05$ ).

**Table 1. The clinical characteristics, biochemical analytes of participants enrolled in the present study.**

Parameters	Group 1 (Healthy Lean Subjects)	Group 2 (Obese Subjects)	P value
n (%)	61 /115 (53.9)	54 /115 (46.1%)	-
Male / Female Ratio	25/36	24/30	-
Age (year)	$34.3 \pm 12$	$34.5 \pm 11$	0.592 NS
Systolic blood pressure (mmHg)	$104.4 \pm 2.5$	$116.4 \pm 3.1$	0.01*
Diastolic blood pressure (mmHg)	$65.1 \pm 1.2$	$70.3 \pm 1.5$	0.03*
Waist Circumference – (WC) (cm)	$74.53 \pm 6.21$	$112.4 \pm 14.2$	$P < 0.001$ ***
Hip (cm)	$97.54 \pm 6.11$	$123.41 \pm 11.27$	$P < 0.001$ **
Waist to Hip Ratio (WHR)	$0.73 \pm 0.05$	$0.94 \pm 0.07$	$P < 0.001$ ***
Weight (kg)	$63.3 \pm 5.3$	$106.1 \pm 20.5$	$P < 0.001$ **
Height (m)	$1.64 \pm 0.8$	$1.62 \pm 0.9$	0.760 NS
BMI (kg/m <sup>2</sup> )	$23.15 \pm 1.44$	$39.80 \pm 7.11$	$P < 0.001$ ***
Serum Glucose (mg/dl)	$87.77 \pm 5.89$	$106.22 \pm 19.14$	0.001**
Serum Insulin (μIU/ml)	$13.56 \pm 3.96$	$31.54 \pm 20.87$	$P < 0.001$ ***
A1c%	$4.78 \pm 0.27$	$5.39 \pm 0.70$	$P < 0.001$ ***
HOMA2-IR	$1.75 \pm 1.2$	$3.85 \pm 1.9$	$P < 0.001$ ***
Triglyceride (mg/dl)	$79.13 \pm 27.64$	$153.73 \pm 56.02$	$P < 0.001$ ***
Total Cholesterol (mg/dl)	$153.85 \pm 31.02$	$197.71 \pm 39.35$	$P < 0.001$ ***
HDL - Cholesterol (mg/dl)	$45.63 \pm 8.04$	$37.31 \pm 10.24$	0.001**
Non -HDL - Cholesterol (mg/dl)	$117.19 \pm 32.64$	$160.41 \pm 40.74$	$P < 0.001$ ***
LDL - Cholesterol (mg/dl)	$101.36 \pm 30.40$	$129.86 \pm 39.91$	$P < 0.001$ ***
TyG index	$4.31 \pm 0.1$	$4.75 \pm 0.3$	$P < 0.001$ ***
AIP	$0.03 \pm 0.08$	$0.22 \pm 0.10$	$P < 0.001$ ***
Omentin (pg/ ml)	$10.15 \pm 5.04$	$15.49 \pm 4.20$	$P < 0.001$ ***

Data are represented as (mean ± SD). BMI: body mass index; A1c%: Hemoglobin A1C; HOMA2-IR: homeostasis model assessment of insulin resistance; HDL-Cholesterol: high density lipoprotein-cholesterol, LDL-Cholesterol: Low density lipoprotein-cholesterol; TyG index: Triglyceride to glucose index; AIP: Atherogenic index of plasma. NS: Non-Significant. Statistical significance considered at \*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\* $p < 0.001$ .

Table.2 illustrates the correlations between omentin and the other parameters included in this study. Omentin levels were positively correlated with WC ( $r = 0.377$ ,  $P < 0.001$ ), WHR ( $r = 0.314$ ,  $P = 0.001$ ), Wt. ( $r = 0.280$ ,  $P = 0.002$ ), BMI ( $r = 0.303$ ,  $P = 0.001$ ), S.Glucose ( $r = 0.211$ ,  $P = 0.023$ ), insulin ( $r = 0.350$ ,  $P < 0.001$ ), A1c ( $r = 0.208$ ,  $P$

$= 0.026$ ), HOMA2-IR ( $r = 0.411$ ,  $P < 0.001$ ), TG ( $r = 0.254$ ,  $P = 0.006$ ), total Chol ( $r = 0.216$ ,  $P = 0.020$ ), LDL-Chol ( $r = 0.204$ ,  $P = 0.029$ ), non-HDL-Chol ( $r = 0.265$ ,  $P = 0.004$ ), TyG index ( $r = 0.245$ ,  $P = 0.008$ ), and AIP ( $r = 0.386$ ,  $P < 0.001$ ). A significant negative correlation was found with HDL-Chol ( $r = -0.266$ ,  $P = 0.004$ ).

**Table 2. Correlations between the parameters and Serum Omentin level in the present study.**

Parameters	(Pearson Correlation Coefficient and <i>P</i> value)
Age (year)	( <i>r</i> = 0.147, <i>P</i> = 0.118) NS
Waist Circumference – (WC) (cm)	( <i>r</i> = 0.377, <i>P</i> < 0.001) ***
Hip (cm)	( <i>r</i> = 0.186, <i>P</i> = 0.076) NS
Waist to Hip Ratio (WHR)	( <i>r</i> = 0.314, <i>P</i> = 0.001) **
Weight (kg)	( <i>r</i> = 0.280, <i>P</i> = 0.002) **
Height (m)	( <i>r</i> = - 0.080, <i>P</i> = 0.395) NS
BMI (kg/m <sup>2</sup> )	( <i>r</i> = 0.303, <i>P</i> = 0.001) **
Serum Glucose (mg/dl)	( <i>r</i> = 0.211, <i>P</i> = 0.023) *
Serum Insulin (μIU/ml)	( <i>r</i> = 0.350, <i>P</i> < 0.001) ***
A1c%	( <i>r</i> = 0.208, <i>P</i> = 0.026) *
HOMA2-IR	( <i>r</i> = 0.411, <i>P</i> < 0.001) ***
Triglyceride (mg/dl)	( <i>r</i> = 0.254, <i>P</i> = 0.006) **
Total Cholesterol (mg/dl)	( <i>r</i> = 0.216, <i>P</i> = 0.020) *
HDL - Cholesterol (mg/dl)	( <i>r</i> = - 0.266, <i>P</i> = 0.004) **
Non-HDL - Cholesterol (mg/dl)	( <i>r</i> = 0.265, <i>P</i> = 0.004) **
LDL - Cholesterol (mg/dl)	( <i>r</i> = 0.204, <i>P</i> = 0.029) *
TyG index	( <i>r</i> = 0.245, <i>P</i> = 0.008) **
AIP	( <i>r</i> = 0.386, <i>P</i> < 0.001) ***

Data are represented as (mean ± SD). BMI: body mass index; A1c%: Hemoglobin A1C; HOMA2-IR: homeostasis model assessment of insulin resistance; HDL–Cholesterol: high density lipoprotein-cholesterol. LDL– Cholesterol: Low density lipoprotein-cholesterol; TyG index, Triglyceride to glucose index; AIP: Atherogenic index of plasma. NS: non- significant.

Statistical significance considered at \* *p* < 0.05, \*\* *p* < 0.01, \*\*\* *p* < 0.001.

Multiple regression analysis was performed with adjustment for potential confounders (Table.3). As a result of the analysis, it was demonstrated that omentin levels were found to be independently associated with s. glucose, total Chol, non-HDL-Chol, TyG index and AIP in total population.

**Table 3. Regression Analysis.**

Parameters	Omentin <sup>a</sup>	
	β-coefficient	Sig.
Waist Circumference (cm)	0.285	0.346
Waist to Hip Ratio (WHR)	0.043	0.821
Weight (kg)	0.578	0.380
BMI (kg/m <sup>2</sup> )	0.247	0.698
Serum Glucose (mg/dl)	0.639	0.004**
Serum Insulin (μIU/ml)	-0.050	0.853
A1c%	0.135	0.151
HOMA2-IR	0.429	0.133
Triglyceride (mg/dl)	0.401	0.142
Total Cholesterol (mg/dl)	0.848	0.039*
HDL - Cholesterol (mg/dl)	0.273	0.146
Non-HDL - Cholesterol (mg/dl)	0.953	0.011*
LDL - Cholesterol (mg/dl)	0.071	0.573
TyG index	0.244	0.009**
AIP	0.984	0.008**

a. Dependent variables (Omentin)

Values with statistical significance considered at \* *p* < 0.05, \*\* *p* < 0.01, \*\*\**p* < 0.001.

## Discussion:

The present study demonstrated the clinical features and biochemical analytes in a group of subjects with obesity and compared to the healthy

lean. Obese group showed elevated levels of omentin as compared with lean group. The results also demonstrated increased levels of s.glucose, s. insulin, HOMA2-IR, lipid profile and the other risk factors including AIP and TyG index. Among the strongest predictors of omentin are glucose, total Chol, non-HDL-Chol, TyG index and AIP. These findings point to the presence of risk factors in obese group even in absence of diabetes and overt chronic diseases.

Previous studies investigated serum omentin levels in patients with diabetes or metabolic syndrome informed inconsistent results. Reduced levels of omentin were reported in the previous studies. These studies explained the reduced level of omentin that may be attributed to impaired glucose regulation and insulin resistance in these patients<sup>1,6,7,13</sup>.

In contrast to the previous studies, our results are in line with the findings of studies<sup>4,14,15</sup>. These studies reported increased levels of omentin in diabetic patients and nondiabetic individuals. Type 2 diabetes, cardiovascular disease, hypertension, and hyperlipidemia are all related to visceral obesity<sup>1</sup>. To better understand the omentin, which is a visceral fat depot specific adipokine in obesity, this study was conducted. Our aim was to learn more about the relationship between omentin levels and biochemical parameters and other obesity risk factors. The discovery of adipokines linked to obesity has a major impact on obesity studies. Since

adipokines have been linked to adipose tissue dysfunction<sup>5</sup>.

Despite the fact that omentin is not currently employed as a diagnostic measure, it should be linked to other biochemical markers and risk indices. In order to recognize obese subjects at risk for diabetes, insulin resistance and other metabolic complications.

The study of Flehming et al.<sup>4</sup> assumed that adipokines secretion are altered due to adipose tissue dysfunction<sup>16</sup>. They revealed a separation of a cluster more closely associated to lipid metabolism markers and a cluster encompassing obesity, insulin resistance, and inflammatory parameters in the subgroup of obese patients with T2D. This shows that a subgroup of people with obesity and T2D has extra changes in their lipid metabolism, which may be reflected or mediated by omentin. This lends credence to the idea that adipose tissue malfunction has a role in obesity-related metabolic disorders<sup>5</sup>.

On explanation of elevated omentin levels Niersmann and colleagues<sup>14</sup> stated that "Higher omentin concentrations in individuals at risk for cardiovascular events may reflect a counterregulatory mechanism. Omentin could be upregulated in response to metabolic and inflammatory stimuli that contribute to atherogenesis, but this appears insufficient to protect against the onset of cardiovascular events. If this were true, associations could be explained by reverse causation because higher biomarker levels would represent early symptoms of cardiovascular disease."

The expected mechanism underlying the high levels of omentin in the present study is the interaction between low grade inflammation that associated with obesity and the existence of higher levels of s. glucose and atherogenic lipid profile. It seems like high level of omentin behave as anti-inflammatory, antiatherogenic and anti-cardiovascular factor in a stage of obesity before the onset of frank insulin resistance state or the prediabetes condition.

A cohort study on non-diabetic participants was conducted by Herder et al.<sup>15</sup> revealed that omentin levels were positively related to the increases in fasting glucose, 2 -hours glucose and hemoglobin A1c and higher baseline omentin levels were associated with increase incident of type 2 diabetes in the study population. "Two possible explanations for the findings from the prospective studies are (i) direct effects of omentin-1 that impair glucose homeostasis and increase diabetes risk or (ii) regulatory mechanisms that involve an upregulation of omentin-1 by proinflammatory and/or metabolic triggers that promote the

development of type 2 diabetes"<sup>15</sup>. Pathogenesis of type 2 diabetes may involve low grade inflammation due to high levels of IL-  $\beta$ , C-reactive protein and certain inflammatory proteins such as inflammasome<sup>17</sup>.

It is interesting that the obese group show impaired fasting s. glucose according to the American Diabetes Association criteria which is more restrict level than world health organization guidelines (fasting serum glucose:100-125 mg/dl consider as prediabetes)<sup>18</sup>. Although the selection of patients was to exclude any previous history for diabetes and other disease. It is apparently obesity has an impact on blood glucose in one way or another. And this may be attributed to high omentin levels of these patients in the present study.

A number of studies have found that diabetic individuals had significantly lower omentin levels than controls in the presence or absence of ischemic heart disease<sup>19</sup>. In another research, diabetic obese individuals with or without cardiovascular disease had significantly lower levels of omentin than healthy controls<sup>20</sup>. The discrepancies which discovered in circulating levels of omentin were thought to be a reflection of metabolic abnormalities in adipose tissue<sup>21</sup>. It was suggested that a compensatory mechanism led to insulin resistance in order to maintain normal glucose levels by a reduction in the response of insulin on adipose and muscle tissues which may result in glucose intolerance. This might play crucial role in the progress of type 2 diabetes<sup>22</sup>.

In contrast to our study, which involved obese adults without diabetes or other chronic conditions, the existence of diabetes was a significant component that associated with low levels of omentin.

It is well established that atherogenic lipid profile components are used as indicators in evaluating atherosclerotic cardiovascular diseases. dyslipidemia is a significant contributor to the progress of coronary heart disease (CHD) associated with obesity<sup>23</sup>. It is clear from the lipid profile in the present study that the obese group had greater levels of triglycerides, total cholesterol, non-HDL – cholesterol, LDL – cholesterol, and lower levels of HDL – cholesterol. These findings are aiming at obesity-related risk factors. This might explain why obese people have higher levels of omentin, which could be a protective mechanism for this adipokine in the context of cardiovascular risk factors. In addition to the presence of regression correlation between omentin total cholesterol, non-HDL-cholesterol, TyG index and AIP may imply the influence of these parameters on the level of omentin.

The experimental investigations suggested that omentin has beneficial effects on cardiometabolic risk<sup>24</sup>. Systemic intake or overexpression of omentin delayed the formation of aortic atherosclerotic lesions and decreased the magnitude of myocardial infarction in mice models suffer from atherosclerosis or ischemia /reperfusion damage, according to preclinical research utilizing animal models<sup>25</sup>.

The atherogenic index of plasma (AIP) is a computed indicator that accurately predicts atherogenicity and atherosclerosis risk in the future. AIP represents the opposing effects of TG and HDL-Chol on inflammation, oxidative stress, extracellular alteration in vascular smooth muscle when the components used to calculate it are taken into account<sup>26</sup>. AIP values of 0.12–0.21 or > 0.21 suggest the prevalence of abdominal obesity, and the integration of WC and AIP may improve the specificity and sensitivity for detecting abdominal obesity in clinical research. The findings show that AIP might be used to measure abdominal obesity<sup>27</sup>. It is necessary to detect cardiovascular risks at subclinical stage. Mostly done using plasma lipoproteins. Studies stated using AIP score as better and easier index to indicate arterial stiffness and atherosclerosis. AIP index was more deteriorated with a combination of obesity and Type2 Diabetes<sup>26</sup>. It is worth to mention the mean value of AIP for obese patients in our study is equal to 0.22.

TyG index is a low-cost clinical indicator of insulin resistance, might be beneficial in identifying those who are at high risk of cardiovascular incidents. researches were showed a positive association between TyG index and Cardiovascular risk factors<sup>28, 29</sup>. The advantages of employing this indicator to identify complications in obese people.

We speculate that Omentin has an impact on the link between variables involved in this study including HOMA2-IR, cholesterol, non-HDL – cholesterol, LDL – cholesterol, AIP score and TyG index in obesity.

This is the first study we are aware of that relates AIP and TyG index with omentin and shows evidence of omentin behavior in obese people.

#### Strengths and limitations:

The inclusion of obese adults without diabetes or overt complications are representative of the patients' group is one of the study's strengths. In addition, the fact that omentin was tested at only one time point is one of the study's limitations, as serial biomarker assessment may allow for more exact estimate. Also, overweight subjects (BMI  $\geq 25$  and  $\leq 30$ ) were not included in the study.

#### Conclusion:

Our findings indicate that serum omentin levels are higher in obese individuals compared to healthy lean subjects. Higher omentin concentrations in obese may reflect a counterregulatory mechanism. Furthermore, omentin was linked to HOMA2-IR and other risk factors indices such as AIP and TyG. Omentin can be used as a metabolic marker in obese.

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#### Authors' declaration:

- Conflicts of Interest: None.
- We hereby confirm that all the Figures and Tables in the manuscript are mine ours. Besides, the Figures and images, which are not mine ours, have been given the permission for re-publication attached with the manuscript.
- Authors sign on ethical consideration's approval.
- Ethical Clearance: The project was approved by the Scientific Unit and Medical Ethics Committee of Alkindy College of Medicine, University of Baghdad at its meeting that numbered 167 and dated at 30<sup>th</sup> of July 2019.

#### Authors' contributions statement:

Conception and Study Design: TE K., AH A. Acquisition of data: AH A., TE K, Data analysis and interpretation: SA M G, TE K. Drafting of the article: TE K. Revision and proofreading: TE K, AH A. Final approval: T E K, AH A, and SA M G

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## الصلة بين مستوى الأومنتين المصلي ومؤشرات مقاومة الانسولين، مستوى الدهون ومؤشرات تصلب الشرايين في المرضى العراقيين المصابين بالسمنة

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### الخلاصة:

الأومنتين-Omentin (أو الأنتلكتين-intelectin) هو أحد المركبات التي تفرز من الدهون الحشوية. يوجد اهتمام بحثي متزايد لربط الأومنتين والسمنة مع عوامل الخطورة. الهدف من هذه الدراسة هو تقييم مستوى الأومنتين في مصل الدم وارتباطه بالمؤشرات الحيوية لمقاومة الأنسولين، مستويات الدهون وبعض عوامل الخطورة المصاحبة للسمنة. أجريت هذه الدراسة المقطعية في وحدة أبحاث وعلاج السمنة - كلية طب الكندي بتطويع (115) فردا (49 من رجال و66 من نساء). تم اختيار الأشخاص الذين تتراوح أعمارهم بين (20-60) عاماً وتصنيفهم إلى مجموعتين وفقاً لمؤشر كتلة الجسم (BMI) المجموعة الأولى (مجموعة السيطرة) اشترك فيها متطوعون أصحاء نحاف 25 رجال / 36 نساء وكتلة الجسم لهم 24.9 - 18.5: مؤشر كتلة الجسم). وتضمنت المجموعة الثانية الأشخاص المصابين بالسمنة 24 رجال / 30 نساء (مؤشر كتلة الجسم  $\leq 30$ ). بينت الدراسة ان المجموعة الثانية (المصابين بالسمنة) لديهم مستويات أومنتين أعلى مقارنة بالمجموعة الأولى للأصحاء (السيطرة)، ( $P < 0.001$ )،  $10.15 \pm 5.04$  pg/ml vs.  $15.49 \pm 4.20$ ). وظهرت المجموعة الثانية مستويات أعلى لمقياس الخصر، نسبة مقياس الخصر الى الورك، الوزن، مؤشر كتلة الجسم وسكر الدم، الانسولين، مؤشر مقاومة الانسولين، مستوى الدهون ومستوى أقل من الكوليسترول الدهني عالي الكثافة (HDL-Cholesterol). الأومنتين ارتبط إيجابيا واحصائيا بالمؤشرات الحيوية كمقياس الخصر، نسبة الخصر الى الورك، الوزن، مؤشر كتلة الجسم، سكر الدم، الانسولين، الهيموغلوبين السكري A1c، مؤشر مقاومة الانسولين، مستويات الكوليسترول منخفض الكثافة، مؤشر الدهون الثلاثية الى السكر (TyG index) triglyceride to glucose index ومؤشر تصلب الشرايين للبلازما (AIP) atherogenic index of plasma ( $P < 0.05$ ). أثبت تحليل الانحدار المتعدد أن مستويات الأومنتين مرتبطة بمستويات سكر الدم، الكوليسترول، مؤشر TyG ومؤشر تصلب الشرايين للبلازما (AIP). تشير النتائج إلى أن مستويات الأومنتين في المصل أعلى في الأفراد المصابين بالسمنة مقارنة بالأشخاص الأصحاء ذوي مؤشر كتلة الجسم الطبيعية. ارتبط الأومنتين أيضا بالمؤشرات الحيوية لمقاومة الأنسولين ومؤشرات عوامل الخطورة مثل مؤشر AIP ومؤشر TyG. يمكن استخدام الأومنتين كعلامة للأبيض في السمنة.

الكلمات المفتاحية: مؤشر تصلب الشرايين في البلازما، مقاومة الانسولين، مستوى الدهون، السمنة، الأومنتين، مؤشر الدهون الثلاثية والسكر في الدم.