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Correlations of Serum Chemerin and Visfatin with other Biochemical Parameters in Iraqi Individuals with Metabolic Syndrome and Type Two Diabetes Mellitus

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Abstract

Chemerin and visfatin are bioactive molecules that regulate numerous physiological functions such as energy equilibrium, insulin action, inflammatory response and vascular homeostasis. The objective of this study is to determine the correlation between serum chemerin and visfatin with other biochemical parameters in Iraqi patients with metabolic syndrome (MetS) and type 2 diabetes mellitus (T2DM). Twenty-two participants with MetS, forty-four with T2DM and twenty-two healthy subjects were recruited in this study. Serum concentrations of chemerin and visfatin, hs-CRP, fasting plasma glucose (FPG), fasting serum insulin (FSI), lipid profile for all participants were measured. Their correlations with the anthropometric parameters, insulin resistance and MetS parameters were specified. The results revealed that the MetS group has the highest serum levels of chemerin and hs-CRP compared to T2DM and the control groups (151.77±10.43 ng/mL, 129.36±5.03 ng/mL and 63.98±14.74ng/mL respectively) with significant difference of (P<0.001) and (8.1±1.1mg/L, 7.96±1.18mg/L and 4.55±2.31mg/L respectively). Visfatin serum concentration was higher in the T2DM group than that of the MetS and control group (63.71±8.30ng/mL, 56.03±10.58 ng/mL and 52.46±14.05 ng/mL respectively). Both of these two adipokines were found to be correlated with some parameters. Moreover, no correlation was found between the two proteins. From the obtained results we concluded that the assessment of chemerin and visfatin levels and their relation to some metabolic parameters can help to identify subjects who are more susceptible to the cardiovascular disease (CVD risk.

Keywords: Chemerin, Visfatin, Metabolic syndrome, T2DM.

1. Introduction

The metabolic syndrome (MetS) refers to a cluster of related metabolic abnormalities, such as central obesity, hypertension, dyslipidemia, hyperglycemia, and insulin resistance (Martínez and Andriantsitohaina, 2017). The central obesity and insulin resistance in particular were known as causative factors for the metabolic syndrome (Srikanthan *et al*., 2016). The increase in the metabolic syndrome prevalence is associated with the increasing overweight, obesity, and physical inactivity (Martínez and Andriantsitohaina, 2017). For the purpose of the diagnosis of MetS, three out of five abnormal conditions should exist in the patient (fasting hyperglycemia, high blood pressure, hypertriglyceridemia, low high-density lipoprotein cholesterol levels, and central obesity) (Alberti *et al.*, 2006). Several studies showed that the MetS is associated with an approximate two-fold increased risk of developing cardiovascular disease (CVD) and a five-fold increased risk for incident type two diabetes mellitus over the next five to ten years (Cornier *et al*., 2008).

Diabetes mellitus type 2 is a combination of disorders that are characterized by elevated blood levels of glucose and are associated with microvascular and macrovascular complications (Zaccardi *et al*., 2015). The endogenous insulin deficiency or resistance to the insulin action in muscle, fat and liver in addition to the inadequate response by the pancreatic beta cells result in hyperglycemia (Wolfs *et al*., 2009). The adipose tissue is defined as an active endocrine organ that secretes an enormous number of bioactive mediators (adipokines) that signal to the organs of metabolic importance including the brain, liver, skeletal muscle, and the immune system thereby modulating hemostasis, blood pressure, glucose and lipid metabolism, inflammation and atherosclerosis (Bozaoglu *et al.*, 2007). These adipokines include: adiponectin, leptin, omentin, resistin, and interleukin-6, tumor necrosis factor-α, visfatin, vaspin and chemerin (Yan *et al.*, 2012).

Chemerin is one of these bioactive mediators defined as a multifunctional peptide involved in the glucose and lipid metabolism. Raised levels of this peptide have been associated with insulin resistance and systemic inflammation (Fatima *et al*., 2015). This protein is highly expressed in liver and the white adipose tissue. Increased

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levels of chemerin have been observed in mice and humans that are obese and have T2DM (Bozaoglu *et al*., 2007). The metabolic effect of chemerin in diabetes is the dysregulation of insulin and glucose metabolism (Lehrke *et al*., 2009).

Visfatin is the adipokine that exerts insulin-mimetic effects, which stimulate muscle and adipocyte glucose transport, and inhibit the hepatocyte glucose creation. It is expressed mainly in the visceral adipose tissues as well as in the human liver, muscles, and macrophages. Visfatin prompts the production of cytokines, such as interleukin-6 and tumor necrosis factor-α, in the human leukocytes. Its plasma level rises during chronic inflammatory conditions such as psoriasis, arthritis, and obesity (Adghate, 2008). Also, it increases in T2DM, which is characterized by insulin resistance (IR), and is usually observed in gestational diabetes mellitus (GDM) (Lewandowski *et al*., 2007). The relations between visfatin and metabolic syndrome, such as insulin resistance and dyslipidemia in humans have recently been studied, and numerous aspects of these relations are unknown. However, Mohammadi *et al.* found that high circulating levels of visfatin could be in healthy relations with cardiovascular risk factors, insulin resistance status and adiponectin in diabetic patients (Mohammadi *et al*., 2011).

The purpose of this study is to determine the serum levels of chemerin and visfatin in Iraqi individuals with the metabolic syndrome and type two diabetic patients. Furthermore, it is designed to find the correlation between these two adipokines; chemerin and visfatin as well as between them and other anthropometric and biochemical parameters.

2. Materials and Methods

2.1. Study Subjects

This cross sectional study was conducted in cooperation with the National Diabetes Center for Treatment and Research at Al-Mustansiriya University in Baghdad city, Republic of Iraq. Eighty-eight subjects were enrolled in this study: forty-four patients with T2DM (twenty-seven males and seventeen females) aged between twenty and seventy years; and BMI of 28.35±3.68 kg/m^2, twenty-two patients with MetS (thirteen males and nine females) aged between twenty and sixty years; BMI of 38.71±6.24 kg/m^2, and twenty-two healthy participants (with no family history of diabetes, high cholesterol, hypertension or other diseases) (fourteen males and eight females) aged between twenty and sixty years; BMI of 24.77±3.48 kg/m^2.

Participants with T2DM were included in the study according to the World Health Organization criteria (Alberti and Zimmet, 1998). The diagnosis of MetS was based on the global consensus of MetS according to the 2005 International Diabetes Federation (Alberti *et al.*, 2006). The patients must have central obesity (BMI is >30kg/m²), and any two out of the rest four factors of MetS diagnosis: 1) Elevated Triglycerides ≥ 150 mg/dL (or specific treatment for this lipid abnormality). 2) Decreased HDL- cholesterol value (< 40 mg/dL in males and < 50 mg/dL in females) or specific treatment for this lipid abnormality. 3) Raised blood pressure; systolic blood pressure ≥ 130 mm Hg or diastolic blood pressure ≥ 85 mmHg or (having been diagnosed with hypertension and were treated). 4) Elevated fasting plasma glucose (FPG; ≥ 110 mg/dl) or have been diagnosed with type II diabetes. The exclusion criteria included patients with type one diabetic mellitus, T2DM taking insulin as hypoglycemic therapy, acromegaly, chronic liver and kidney diseases. The study was approved by the Human Research Ethics Committee of the Center, and informed agreements were obtained from each patient.

2.2. Blood Sample Collection

Ten milliliters of venous blood were collected after ten-twelve hours of fasting from each subject and then were then divided into two aliquots. For the first aliquot (2 ml), EDTA containing tube was used for the assessment of fasting plasma glucose, while for the second aliquot (8 ml), biochemistry tubes with a gel separator were used. After thirty minutes of an incubation period, the samples were centrifuged (at 1500 × g for fifteen minutes). A portion of the obtained serum was used for the estimation of lipid profile and uric acid. The second portion of the serum used for the subsequent assay of fasting serum insulin, hs-CRP, chemerin and visfatin was stored at -20^{o}C.

2.3. Anthropometric Measurements

Weight (Kg), height (cm), and waist circumference (cm) were measured for all the participants. The body mass index (BMI) was calculated by dividing the weight (in kg) over the height square (m^2), waist to height ratio (WHtR) as well as body fat percentage (BF %) was calculated according to the following equations:

WHtR = waist (cm)/ height (cm)

BF% = (1.20 x BMI) + (0.23 x age) - (10.8 x sex) - 5.4 …. (Deurenberg *et al*., 1991)

Sex: male=1, female=2

2.4. Blood Pressure Measurements

Mercury sphygmomanometer was used to measure the Systolic blood pressure (SBP) and diastolic blood pressure (DBP) (mmHg) in a sitting position. The mean arterial pressure (MAP) was calculated from these measurements according to the equation below:

MAP = DBP + (SBP-DBP)/3 …… (Bouchra *et al*., 2005)

2.5. Laboratory Tests

The estimation of plasma glucose was performed by a glucose oxidase method. The total serum cholesterol was estimated by enzymatic colorimetric tests with cholesterol esterase and cholesterol oxidase, while the serum triglycerides evaluation was done by the enzymatic colorimetric tests with glycerol phosphate oxidase. The HDL-cholesterol was evaluated after precipitation of the apolipoprotein B-containing lipoproteins with phosphotungstic acid. The low-density lipoprotein cholesterol was calculated by the Friedewald formula (Friedewald *et al*., 1972). The atherogenic index of plasma (AIP) was calculated using the equation:

AIP =Log (Tg / HDL-C) ….. (Dobiášová and Frohlich, 2001)

The homeostasis model assessment of insulin resistance (HOMA-IR) was calculated from fasting insulin and glucose by the equation bellow (Mattews *et al*., 1985):

HOMA-IR = insulin (micro units per milliliter) × glucose (mg/dl) / 405

The insulin resistance was defined at the cutoff point corresponding to HOMA-IR ≥ 3.8 (Shirai, 2004). The fasting serum insulin level was estimated by ELISA using the commercially available ELISA kit (Monobind Inc., U.S.A). High sensitivity C-reactive protein was measured by bioactive diagnostic (Germany) ELISA kit. Human chemerin serum level was measured using the commercially available chemerin ELISA assays kit (Ray Biotechnology Company, U.S.A). The visfatin serum level was estimated also by using Ray Bio Visfatin Enzyme Immunoassay (EIA) Kit (U.S.A). All ELISA procedures were carried out according to the manufacturer's instructions.

2.6. Statistical Analysis

In order to analyze the data statistically, the IBM SPSS software package (version 22.0) was used. The variables were reported as means ± standard deviation. The groups were compared by using one way ANOVA and post hoc Tukey test. The correlations between the serum of visfatin, chemerin and other variables were detected using the Pearson's correlation analysis, with a *P* value of <0.05 indicating the statistically significant difference.

3. Result

Our study consisted of eighty-eight participants divided into three groups: T2DM group (n=44), MetS group (n=22) and the control group (n=22). The general anthropometric, clinical and biochemical features of the participants are represented in Table 1. The Statistical ANOVA and post hoc test showed that there is a significant difference in age between the "control and T2DM" groups as well as "T2DM and MetS" groups at ($P < 0.001$), but there wasn't any significant difference between the control and MetS groups. Waist circumference & waist to height ratio showed that there was a high significant difference in T2DM and MetS groups compared with the control group at ($P < 0.001$), while there wasn't any significant difference in the comparison of T2DM & MetS groups together. BMI was significantly higher in the metabolic syndrome group and T2DM group compared to the control group at ($P < 0.001$), as well as BF %.

Fasting plasma glucose (FPG) in the T2DM group was significantly higher than Mets and control groups at ($P < 0.001$), while FSI and HOMA-IR in Mets group were significantly higher than T2DM and control groups at ($P < 0.001$). By comparing the means of total serum cholesterol of the three groups, it is found that there were no significant differences between them.

Table 1. Clinical and biochemical characteristics of the study

Factor	Control N=22	T2DM N=44	MetSN=22	*P*
Age (y)	33.41±9.55	56.68±8.24 $^{**a, c}$	37.59±10.15 $^{** c}$	<0.001
Sex (M/F)	14M/8F	27M/17F	13M/9F	____
WC (cm)	82.27±7.80	97.64±14.63 ^{**a}	103.0±10.57 ^{**b}	<0.001
BMI(kg/m^2)	24.77±3.48	28.35±3.68 $^{**a, c}$	38.71±6.24 $^{**b, c}$	<0.001
BF%	25.05±7.56	35.23±7.84 $^{**a, c}$	43.16±9.02 $^{**b, c}$	<0.001
WHtR	0.48±0.03	0.58±0.09 ^{**a}	0.62±0.06 ^{**b}	<0.001
FPG(mg/dL)	87.95±10.81	171.09±53.76 ^{**a,c}	109.64±7.16 $^{** c}$	<0.001
FSI(μlU/mL)	7.55±1.86	13.13±1.92 ^{**a,c}	19.25±1.09 $^{**b, c}$	<0.001
HOMA-IR (%)	0.97±0.25	1.96±0.34 ^{**a,c}	2.56±0.16 $^{**b, c}$	<0.001
TC(mg/dL)	168.90±36.96	181.70±43.84	128.27±33.13	0.259
TG(mg/dL)	77.18±29.26	148.59±93.32 ^{**a,c}	84.31±40.40 $^{** c}$	0.001
HDL-C(mg/dL)	47.38±12.09	37.38±9.86 $^{**a, *c}$	44±8.83 ^{*c}	0.001
LDL-C(mg/dL)	106.06±38.14	116±39.78	128±32.29	0.160
VLDL-C(mg/dL)	15.02±6.31	29.70±18.72 ^{**a,c}	16.86±7.90 $^{** c}$	0.001
SBP(mmHg)	118.9±16.3	141.8±23.5 ^{**a,c}	125±19.5 $^{** c}$	<0.001
DBP(mmHg)	74.5±10.7	81.5±13.8	74.09±11.6	0.030
MAP(mmHg)	89.3±11.5	101.6±16.2 ^{**a,*c}	91±13.8 $^{* c}$	0.002
AIP	0.11±0.05	0.55±0.28 ^{**a,c}	0.26±0.19 $^{** c}$	<0.001
hs-CRP(mg/L)	4.55±2.31	7.96±1.18 ^{**a}	8.1±1.1 ^{**b}	<0.001
Chemerin(ng/mL)	63.98±14.74	129.36±5.03 ^{**a,c}	151.77±10.43 ^{**b,c}	<0.001
Visfatin(ng/mL)	52.46±14.05	63.71±8.30 ^{**a,*c}	56.03±10.58 $^{* c}$	<0.001

Results were expressed as mean ± SD, ANOVA test was used for the purpose of comparison between the three groups. *P< 0.05 is significant, **P<0.01 is highly significant. a refers to the significant differences between control and T2DM. b refers to the significant differences between controls and MetS. c refers to the significant differences between for T2DM and MetS. T2DM =Type 2 diabetes mellitus, MetS=Metabolic syndrome, WC= waist circumference, BMI=body mass index, BF %=body fat percentage, WHtR=waist to height ratio, FPG=fasting plasma glucose, HOMA-IR= homeostasis model of assessment-insulin resistance, TC=total cholesterol, TG= triglycerides, HDL= high-density lipoprotein, LDL= low-density lipoprotein; VLDL= very low-density lipoprotein, SBP= systolic blood pressure, DBP=diastolic blood pressure, MAP= mean arterial pressure, AIP= atherogenic index of plasma, hs-CRP=high-sensitivity C-reactive protein.

Serum TG in the T2DM group was significantly higher compared to that of both the control and the metabolic syndrome groups ($P < 0.01$), as well as VLDL-C, while mean of serum HDL-C was significantly higher in the control group compared to the patients groups ($P< 0.01$). However, the mean of serum LDL-C was higher in the MetS group compared to the T2DM and control group. The means of Systolic blood pressure (SBP), diastolic blood pressure (DBP), arterial pressure (MAP) and atherogenic index of plasma (AIP) revealed a significant increase in the diabetic group compared with the metabolic syndrome and control groups. Figure 1 illustrates that chemerin serum levels in the MetS group were higher than in the T2DM and control group, as well as hs-CRP (Figure 2). In contrast, visfatin serum levels in the T2DM group were higher than those of the Mets and control groups, Figure (3).

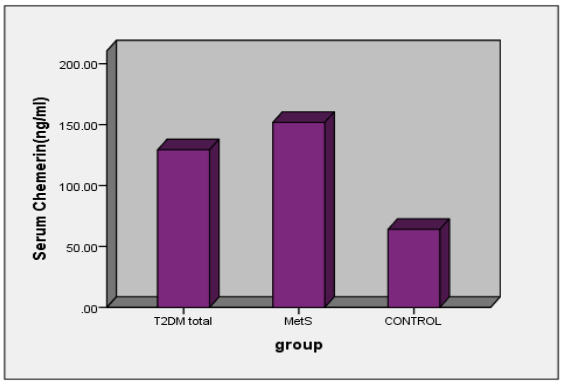

Figure 1. Chemerin serum levels of the three groups

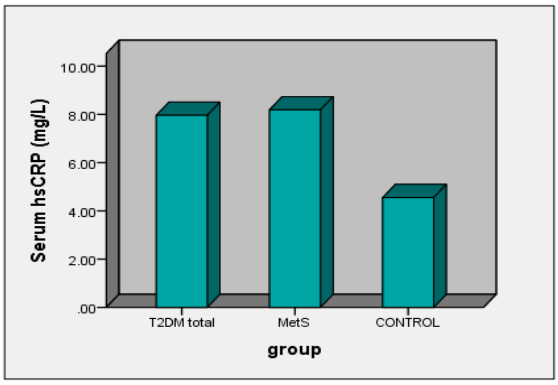

Figure 2. hs-CRP serum Levels of the three groups

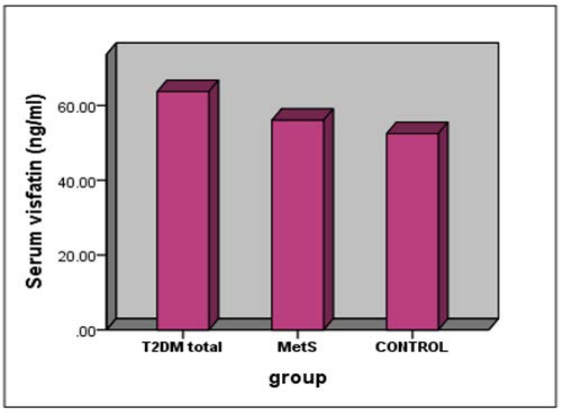

Figure 3: Visfatin serum levels of the three groups

Table 2 shows the Pearson correlation coefficient of serum chemerin and serum visfatin in the diabetic group. Based on the results, the chemerin serum level had a positive correlation with BMI (P =0.011), BF% (P = 0.049), WHtR (P = 0.036) and hs-CRP (P = 0.002), while the visfatin serum level had a positive correlation with BF % (P = 0.041), and is negatively correlated with triglycerides (P=< 0.001), VLDL (P=< 0.001) and AIP (P = 0.01). According to Table 3, chemerin serum levels in the MetS group had a positive correlation with gender (P = 0.001), BF % (P= 0.032), triglycerides (P= 0.001), VLDL (P=0.001) and AIP (P= 0.004). Visfatin serum levels were positively correlated with BMI (P= 0.011), BF % (P= 0.049) and WHtR (P= 0.036), while being negatively correlated with gender (P = 0.038).

Table 2. Correlations between chemerin and visfatin serum levels with anthropometric and laboratory data of T2DM group (n=44)

Parameter	Serum Chemerin		Serum visfatin	
	R	*P*	*R*	*P*
Age(y)	0.055	0.723	0.242	0.114
Sex (M/F)	0.102	0.510	0.251	0.101
WC(cm)	0.211	0.180	-0.172	0.256
BMI	0.380	0.011	0.143	0.353
BF%	0.299	0.049	0.310	0.041
WHtR	0.318	0.036	-0.103	0.507
FPG(mg/dL)	0.047	0.761	0.022	0.888
Insulin(µlU/mL)	0.215	0.162	0.293	0.054
HOMA-IR (%)	0.189	0.219	0.239	0.119
TC(mg/dL)	0.147	0.342	-0.157	0.307
TG(mg/dL)	0.085	0.584	-0.509	< 0.001
HDL(mg/dL)	0.098	0.525	0.182	0.238
LDL(mg/dL)	0.094	0.544	0.020	0.895
VLDL(mg/dL)	0.086	0.578	-0.507	< 0.001
SBP(mmHg)	0.139	0.369	0.003	0.987
DBP(mmHg)	-0.030	0.849	-0.041	0.790
MAP(mmHg)	0.050	0.746	-0.022	0.886
AIP	0.044	0.779	-0.382	0.01
hs-CRP(mg/L)	0.449	0.002	0.294	0.053
Chemerin(ng/mL)	1	-	0.066	0.673
Visfatin(ng/mL)	0.066	0.673	1	-

r, Pearson coefficient. *Statistically significant at $P \leqslant 0.05$. **highly significant at $P \leqslant 0.01$. WC= waist circumference, BMI=body mass index, BF%=body fat percentage, WHtR=waist to height ratio, FPG=fasting plasma glucose, HOMA-IR= homeostasis model of assessment-insulin resistance, TC=total cholesterol, TG= triglycerides, HDL= high-density lipoprotein, LDL= low-density lipoprotein, VLDL= very low-density lipoprotein, SBP= systolic blood pressure, DBP=diastolic blood pressure, MAP= mean arterial pressure, AIP= atherogenic index of plasma, hs-CRP=high-sensitivity C-reactive protein.

Table 3. Correlations between chemerin and visfatin serum levels and anthropometric and laboratory data of MetS group (n=22)

Parameter	Serum Chemerin		Serum Visfatin	
	R	*P*	*R*	*P*
Age(y)	0.080	0.725	0.038	0.868
Sex(M/F)	0.648	0.001	-0.445	0.038
Waist(cm)	0.160	0.478	0.211	0.180
BMI	0.060	0.790	0.380	0.011
BF%	0.458	0.032	0.299	0.049
WHtR	0.333	0.130	0.318	0.036
FPG(mg/dL)	-0.003	0.991	-0.077	0.733
Insulin(μlU/mL)	-0.132	0.558	-0.238	0.287
HOMA-IR(%)	-0.110	0.625	-0.222	0.320
TC(mg/dL)	0.388	0.075	0.147	0.342
TG(mg/dL)	0.672	0.001	0.085	0.584
HDL(mg/dL)	0.093	0.680	0.098	0.525
LDL(mg/dL)	0.199	0.374	0.094	0.544
VLDL(mg/dL)	0.661	0.001	0.086	0.578
SBP(mmHg)	-0.042	0.852	0.025	0.913
DBP(mmHg)	-0.235	0.293	0.021	0.927
MAP(mmHg)	-0.151	0.501	0.023	0.918
AIP	0.587	0.004	0.044	0.779
hs-CRP(mg/L)	-0.011	0.961	0.233	0.297
Chemerin (ng/mL)	1	-	-0.364	0.096
Visfatin (ng/mL)	-0.364	0.096	1	-

r, Pearson coefficient. *Statistically significant at $P \leqslant 0.05$. **highly significant at $P \leqslant 0.01$. WC= waist circumference, BMI=body mass index, BF%=body fat percentage, WHtR=waist to height ratio, FPG=fasting plasma glucose, HOMA-IR= homeostasis model of assessment-insulin resistance, TC=total cholesterol, TG= triglycerides, HDL= high-density lipoprotein; LDL= low-density lipoprotein; VLDL= very low-density lipoprotein, SBP= systolic blood pressure, DBP=diastolic blood pressure, MAP= mean arterial pressure, AIP= atherogenic index of plasma, hs-CRP=high-sensitivity C-reactive protein.

4. Discussion

The present study aimed to assess the levels of serum chemerin and visfatin in patients with T2DM and MetS. It is aimed also to examine the correlations between these two adipokines. The results of the study reveal that serum chemerin levels were higher in the subjects with MetS than those of the diabetic patients and healthy individuals. In contrast, serum visfatin levels in the T2DM group were higher than the Mets and control groups. The statistical tests showed that there was no correlation between chemerin and visfatin among the studied groups. Previous studies revealed that the serum level of chemerin is higher in individuals with MetS compared to healthy individuals (Jialal *et al.*, 2013, Chu *et al*., 2012 and Bozaoglu *et al.*, 2009). In addition, Bozaoglu *et al*. found that the serum level of chemerin was associated with many indicators of this syndrome such as triglyceride, HDL, and fasting insulin level among a Mexican-American population. They also found that the level of this adipokine was higher in obese than lean individuals (Bozaoglu *et al*., 2009). It is well- known that obesity is one of the most important outcomes of modern lifestyles which continually raise the risk for many diseases development.

BMI and waist circumference, body fat percentage are markers for obesity. As obesity is accompanied with increased body fat and this adipokine is produced by adipose tissues, the increase in cell count and adipose tissue results in an increase in the production of this adipokine (Zanganeh *et al*., 2016). Numerous studies indicate that chemerin affects glucose homeostasis, and could be the link between increased adipose tissue mass/fatty liver disease and obesity-related metabolic and inflammatory diseases. Chemerin was found to be associated with many components of the MetS, including BMI, triglycerides, high-density lipoprotein cholesterol, and hypertension, and also with systemic markers of inflammation, such as high sensitivity C-reactive protein (hs-CRP), interleukin-6 (Chakaroun *et al*., 2012). These results are in agreement with the results of the current study which showed a positive correlation between chemerin and some of the MetS parameters in both patient groups, such as BF %, BMI, triglycerides, VLDL, hs-CRP and AIP (Tables 2 & 3). As illustrated in Figure 1, serum levels of chemerin in the T2DM patients were higher compared to the healthy subjects and such result came in concordance with that of El-Mesallamy et al. and Tarik et al. (El-Mesallamy *et al*., 2011 and Tarek *et al*., 2013), who found that chemerin serum levels in patients with type two diabetics were higher than that in non-diabetic healthy individuals. Similarly, Susana et al. found that circulating chemerin concentrations were elevated in diabetic patients (Susana *et al.*, 2014).

Visfatin is the adipokine that has insulin-mimetic effects and stimulates the muscle and adipocyte glucose transport, and inhibits the hepatocyte glucose creation (Samiha *et al.*, 2013). Diabetic patients have higher levels of serum visfatin compared to MetS and healthy individuals, as shown in Figure 3. Increased visfatin levels in the T2DM patients are independent on obesity and insulin resistance and are mainly determined by levels of fasting glucose and triglycerides (Esteghamati *et al*., 2011). Previous studies found that visfatin levels were higher in patients with T2DM compared to controls (Dogru *et al*., 2007 and Samiha *et al*., 2013). This study revealed that visfatin, in the T2DM group, was not correlated with BMI and WHtR, whereas it was correlated positively with BF% and negatively with TG, AIP and VLDL. In the patients with MetS, visfatin appeared to be correlated positively with BMI and WHtR, and was negatively correlated with sex, disimilar to chemerin which showed a positive correlation with sex as illustrated in Tables 2 & 3 respectively. Both of these adipokines have a positive correlation with body fat percentage. However, in the MetS group, chemerin had a positive correlation with TG, VLDL and AIP.

Many studies showed that there is a strong association between obesity and the metabolic syndrome so that the accumulation of visceral fat has an essential role in metabolic syndrome, cardiovascular disease and obesity-related disorders such as diabetes mellitus, hyperlipidemia and hypertension. The secretion of numerous adipokines by the adipose tissue may be the major mechanisms in these lifestyle-related diseases (Matsuzawa, 2006). Ahmed et al. found that visfatin may be implicated in diabetes pathogenesis, and plays an important role in the development of metabolic syndrome (Ahmed *et al.*, 2015). Recent studies showed that the measurement of serum

levels of chemerin can be effective in the diagnosis of metabolic syndrome (Zanganeh *et al.*, 2016). Lachine et al. concluded that serum level of chemerin correlates with cardio-metabolic disease, with a significant association between chemerin serum concentration and the severity of CAD in Egyptian patients with T2DM (Lachine *et al*., 2016).

5. Conclusion

From the obtained results we conclud that visfatin and chemerin may contribute to the development of insulin resistance and diabetes mellitus. Furthermore, the assessment of chemerin and visfatin levels and their correlations with some metabolic syndrome parameters can help to identify subjects who are most susceptible to the CVD risk.

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