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The Clinical Significance of Vaspin and Chemerin in Diabetic Obese Libyan Patients

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ABSTRACT

Recently, vaspin and chemerin have been identified as interesting novel adipokines having insulin-sensitizing effects. However, the relationship between them has not been elucidated; and their circulating levels in type 2 diabetes mellitus (T2DM) have not been adequately studied. Therefore, this study was designed to investigate whether their levels are altered in Libyan T2DM patients and to study the correlation of these novel adipokines with each other and with insulin resistance, body mass index (BMI) and lipid profile. After an overnight fasting, a single blood sample was obtained. The levels of vaspin, chemerin, and insulin were measured in non obese and obese T2DM patients together with matched healthy non diabetic control subjects by enzyme-linked immunosorbent assay (ELISA), whereas glucose and lipid profile were measured using colorimetric enzymatic methods. Obesity was assessed using BMI and insulin resistance was measured using Homeostasis Model Assessment-Insulin Resistance (HOMA-IR).Vaspin and chemerin levels were found to be significantly elevated in non obese (1.0± 0.50 and 116.5 ± 5.6 ng/ml, respectively) and obese T2DM patients (2.70 ± 0.50 and 174.11 ± 8.60 ng/ml, respectively) compared with control subjects (0.3 ± 0.1 and 78.2 ± 9.4 ng/ml, respectively) at $P < 0.01$. In addition, vaspin and chemerin levels were found to be significantly correlated with each other and with BMI, insulin resistance, and lipid profile. In conclusion, both vaspin and chemerin might play an important role in the pathogenesis of T2DM. In addition, those adipokines (vaspin and chemerin) are significantly interrelated with each other.

Key words: Adipokines, vaspin, chemerin, type 2 diabetes mellitus, obesity

Introduction

Recently, there has been a worldwide increase in the incidence of obesity associated with a metabolic syndrome known as type 2 diabetes (Butler, 2004); and predicted estimates suggest that the population with this syndrome may double to ≈300 million by the year 2025 (Zimmet, 2003). One of the critical determinants for the development of this obesity may be an increase in the regional distribution of body fat, i.e., abdominal obesity. The latter often shows clustering of atherogenic risk factors i.e., hypertension, dyslipidemia, alterations in coagulation and inflammatory cytokine profiles, and hyperinsulinemia and insulin resistance (Seida *et al.,* 2003).

When weight is gained, hyperplasia and hypertrophy of adipocytes within adipose tissue are found. In recent years it could be demonstrated convincingly that fat cells differentially secrete various proteins, so-called adipokines, which link obesity with components of the metabolic syndrome (Trujillo and Scherer, 2006).

Hida *et al.* (2005) characterized vaspin as an interesting novel adipokine with insulin-sensitizing effects. Vaspin belongs to the serine protease inhibitor (serpine) superfamily and is produced in the visceral adipose tissue depot of Otsuka Long-Evans Tokushima Fatty (OLETF) rats, an animal model of obesity with T2DM.

Kloting *et al.* (2006) postulated that induction of vaspin mRNA expression in human adipose tissue could represent a compensatory mechanism associated with obesity, severe insulin resistance, and type 2 diabetes. Thus, the adipokine vaspin is a novel candidate to link human obesity to its related metabolic alterations.

Chemerin was recently found to be an adipokine, and its expression was increased in states of obesity (Bozaoglu *et al.,* 2007).

Chemerin mRNA expression was increased in adipose tissue of obese and type 2 diabetic animals, it was predominantly expressed by adipocytes in adipose tissue, and was significantly induced upon differentiation of 3T3-L1 cells into mature adipocytes (Takahashi *et al.,* 2008).

A growing body of human experimental data indicates that serum chemerin levels are elevated in patients with obesity and that they exhibit a positive correlation with various aspects of the metabolic syndrome. Thus, the role of chemerin in metabolism might provide a link between obesity and obesity related disorders such as type 2 diabetes (Ernst and Sinal, 2010).

Aim of the study:

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The present study aimed to investigate the role of both vaspin and chemerin in the pathogenesis of type 2 diabetes mellitus and the relationship between the two adipokines. Also, the relation of both adipokines with insulin resistance, BMI and lipid profile in the studied groups.

Subjects And Methods

- The current study was conducted on 60 type 2 diabetic patients. They were subdivided according to BMI into 30 obese (BMI > 25 kg/m^2) patients (22 females and 8 males) and 30 non obese (BMI < 25 kg/m^2) patients (19 females and 11 males) with mean of age 58.0 and 57.3 years respectively.
- The patients were chosen randomly from those attending the outpatient Clinic of Tripoli university hospital.
- Complete clinical examination was done for all patients to exclude any diabetic complication such as neurologic, cardiac, kidney, and eye complications.
- Twenty healthy subjects matching the average age and socioeconomic status were selected randomly as a control group. They were subdivided to ten obese (BMI > 25 kg/m^2) subjects (7 females and 3 males) and ten non obese (BMI < 25 kg/m^2) subjects (6 females and 4 males).
- The healthy subjects were chosen randomly from those working at Tripoli university hospital.
- Complete clinical examination was also done for all healthy subjects to exclude any health problem that may they suffer from.
- Weight and height were measured for each subject then the body mass index was calculated as following: BMI = Body weight in Kg / (height in m^2) = Kg/m^2 (Bray, 1987).

Samples and laboratory analysis:

A sample of 10 ml venous blood was collected from each subject after an overnight fasting. The venous blood sample was divided into three test tubes. 1 ml was added to a mixture of potassium oxalate and sodium fluoride (for plasma glucose estimation by oxidase/peroxidase kit) (Caraway and Watts, 1987), 2 ml was added to EDTA powder (whole blood to estimate HbA1c by a direct enzymatic method) (Goldstein *et al*., 2004), and the remaining 7 ml were allowed to clot at room temperature then centrifuged at 1000 rpm for 15 minutes. Serum was separated and divided into aliquots then frozen at -20 ^{0}C till the time of assay.

The serum samples were used to estimate the following parameters:

1- Insulin: by a solid phase enzyme linked immunosorbent assay (ELISA) Kit (Andersen *et al*., 1993).

2- Triglycerides: by enzymatic colorimetric kit (Fossati and Principe, 1982).

3- Total cholesterol: by enzymatic colorimetric kit (Allain *et al*., 1974).

4- HDL-cholesterol: by phosphotungstate precipitation kit (Lopes-Virella *et al*., 1977).

5- Vaspin and Chemerin: by a solid phase enzyme linked immunosorbent assay (ELISA) Kit (Porstmann and Kiessig, 1992).

- LDL-cholesterol was estimated by the equation of Friedewald *et al*. (1972) LDL-Cholesterol=Total cholesterol - (triglyceride/5 + HDL-Cholesterol).
- Insulin resistance was estimated by Homeostasis Model Assessment (Matthews *et al*., 1985 and Levy *et al*., 1988).
- HOMA-R = $\dfrac{[\text{fasting insulin (}\mu\text{IU/ml) X fasting glucose (mmol/L)}]}{22.5}$

Statistical Methods:

Statistical Package of social science (SPSS) version 9.0 was used for analysis of data. Data was summarized as mean ± SD. T test was used for analysis of data.

Results:

In the diabetic group there was a highly significant increase in FBG, HbA1c, T-cholesterol, TG, HDL-c, LDL-c, vaspin, chemerin, insulin, and HOMA compared to the control group whereas the BMI was not significantly increased compared to the control group (Table 1).

Table 1: Comparison between the mean of different parameters in type 2 diabetic patients and controls

Variables	Type 2 diabetic patients	Control	P-value
	Mean ± SD	Mean ± SD	
Age (years)	57.7 ± 3.5	58.0 ± 3.3	0.8
FBG (mg/dl)	110.4 ± 13.4	84. 9 ± 8.4	0.0001*
BMI(kg/m^2)	33.6 ± 10.3	33.0 ± 10.5	0.8
HbA1c (%)	8.3 ± 0.8	5.5 ± 0.6	0.0001*

T-Cholesterol (mg/dl)	223.5 ± 27.2	178.7 ± 6.8	0.0001*
TG (mg/dl)	162.2 ± 30.3	88.7 ± 8.0	0.0001*
HDL-c (mg/ dl)	41.9 ± 7.1	48.3 ± 6.0	0.0001*
LDL-c (mg/dl)	149.3 ± 26.9	112.4 ± 8.7	0.0001*
Vaspin (ng/ml)	1.8 ± 0.5	0.3 ±0.08	0.0001*
Chemerin (ng/ml)	145.3 ± 29.9	78.2 ± 9.4	0.0001*
Insulin (µU/ml)	48.8 ± 27.8	6.4 ± 1.0	0.0001*
HOMA	14.0 ± 9.0	1.4 ± 0.3	0.0001*

- P value < 0.05 is considered to be significant.

In obese type 2 diabetic group, there was a significant increase in FBG and HbA1c compared to obese control group. T-cholesterol, TG, HDL-c, LDL-c, vaspin, chemerin, insulin, and HOMA were highly significantly increased compared to obese control group whereas BMI was not significantly increased compared to obese control group (Table 2).

Table 2: Comparison between the mean of different parameters in obese type 2 diabetic patients and obese controls

Variables	Obese type 2 diabetic patients	Obese control	P-value
	Mean ± SD	Mean ± SD	
Age (years)	58.0 ± 3.5	58.5 ± 4.0	0.7
FBG (mg/dl)	120.7 ± 8.4	83.3 ± 8.6	0.001*
BMI(kg/m^2)	43.0 ± 5.6	43.0 ± 2.6	0.9
HbA1c (%)	8.7 ± 0.8	5.6 ± 0.5	0.001*
T-Cholesterol(mg/dl)	247.6 ± 14.8	178.4 ± 7.1	0.0001*
TG (mg/dl)	187.6 ± 15.2	87.7 ± 8.3	0.0001*
HDL-c (mg/ dl)	37.0 ± 2.9	52.0 ± 4.5	0.0001*
LDL-c (mg/dl)	173.4 ± 12.7	108.9 ± 7.1	0.0001*
Vaspin (ng/ml)	2.7 ± 0 .5	0.3 ± 0.08	0.0001*
Chemerin (ng/ml)	174.1 ± 8.6	86.2 ± 4.3	0.0001*
Insulin (µU/ml)	75.7 ± 8.9	6.5 ± 1.1	0.0001*
HOMA	22.6 ± 3.5	1.4 ± 0.4	0.0001*

- P value < 0.05 is considered to be significant.

In non-obese type 2 diabetic group, there was a highly significant increase in FBG, HbA1c, T-cholesterol, TG, LDL-c, vaspin, chemerin, insulin, and HOMA compared to non-obese control group whereas BMI and HDL-c were not significantly increased compared to non-obese control group (Table 3).

Table 3: Comparison between the mean of different parameters in non-obese type 2 diabetic patients and non-obese controls

Variables	Non Obese type 2 diabetic patients	Non obese control	P-value
	Mean ± SD	Mean ± SD	
Age (years)	57.3 ± 3.4	57.4 ± 2.6	0.9
FBG (mg/dl)	100.1 ± 8.7	86.4 ± 8.3	0.0001*
BMI(kg/m^2)	24.2 ± 1.6	23.0 ± 0.9	0.02*
HbA1c (%)	8.0 ± 0.6	5.3 ± 0.6	0.0001*
T-Cholesterol(mg/dl)	199.4 ± 9.3	179.1 ± 6.8	0.0001*
TG (mg/dl)	136.7 ± 17.3	89.7 ± 7.5	0.0001*
HDL-c (mg/ dl)	46.9 ± 6.5	44.6 ± 4.9	0.3
LDL-c (mg/dl)	125.3 ± 10.5	115.9 ± 9.0	0.01*
Vaspin (ng/ml)	1.0 ± 0.3	0.213 ± 0.07	0.0001*
Chemerin (ng/ml)	116.5 ± 5.6	70.2 ± 5.0	0.0001*
Insulin (µU/ml)	21.9 ± 2.2	6.4 ± 0.9	0.0001*
HOMA	5.4 ± 0.8	1.4 ± 0.3	0.0001*

- P value < 0.05 is considered to be significant.

In obese type 2 diabetic group, there was a highly significant increase in FBG, BMI, HbA1c, T-cholesterol, TG, HDL-c, LDL-c, vaspin, chemerin, insulin, and HOMA compared to non-obese type 2 diabetic group (Table 4).

Table 4: Comparison between the mean of different parameters in obese and non-obese type 2 diabetic patients

Variables	Obese type 2 diabetic patients	Non obese type 2 diabetic patients	P-value
	Mean ± SD	Mean ± SD	
Age (years)	58.0 ± 3.5	57.3 ± 3.4	0.4
FBG (mg/dl)	120.7 ± 8.4	100.1 ± 8.7	0.0001*
BMI(kg/m^2)	43.0 ± 5.6	24.2 ± 1.6	0.0001*
HbA1c (%)	8.7 ± 0.8	8.0 ± 0.6	0.0001*
T-cholesterol(mg/dl)	247.6 ± 14.8	199.4 ± 9.3	0.0001*
TG (mg/dl)	187.6 ± 15.2	136.7 ± 17.3	0.0001*
HDL-c (mg/ dl)	37.0 ± 2.9	46.9 ± 6.5	0.0001*
LDL-c (mg/dl)	173.4 ± 12.7	125.3 ± 10.5	0.0001*

Vaspin (ng/ml)	2.7 ± 0.5	1.0 ± 0.3	0.0001*
Chemerin (ng/ml)	174.1 ± 8.6	116.5 ± 5.6	0.0001*
Insulin (µU/ml)	75.7 ± 8.9	21.9 ± 2.2	0.0001*
HOMA	22.6 ± 3.5	5.4 ± 0.8	0.0001*

- P value < 0.05 is considered to be significant.

There was a significant positive correlation between vaspin, chemerin, insulin, and HOMA with each other and with the other studied parameters except with HDL-c, the correlation was significantly negative in type 2 diabetic group (Table 5).

Table 5: Correlation between vaspin, chemerin, insulin and HOMA in type 2 diabetic patients and other studied parameters

Variables		VASPIN	CHEMERIN	INSULIN	HOMA
Vaspin (ng/ml)	r		0.9	0.8	0.8
	P-value		.0001*	.0001*	.0001*
Chemerin(ng/ml)	r	0.9		0.9	0.9
	P-value	.0001*		.0001*	.0001*
Insulin (µU/ml)	r	0.8	0.9		1.0
	P-value	.0001*	.0001*		.0001*
HOMA	r	0.8	0.9	1.0	
	P-value	.0001*	.0001*	.0001*	
FBG (mg/dl)	r	0.7	0.8	0.8	0.9
	P-value	.0001*	.0001*	.0001*	.0001*
BMI (kg/m^2)	r	0.9	1.0	0.9	0.9
	P-value	.0001*	.0001*	.0001*	.0001*
HbA1c (%)	r	0.5	0.5	0.4	0.4
	P-value	.0001*	.0001*	.0001*	.0001*
T-cholesterol (mg/dl)	r	0.8	0.9	0.9	0.8
	P-value	.0001*	.0001*	.0001*	.0001*
TG (mg/dl)	r	0.8	0.8	0.8	0.8
	P-value	.0001*	.0001*	.0001*	.0001*
HDL-c (mg/dl)	r	- 0.7	- 0.7	- 0.7	- 0.7
	P-value	.0001*	.0001*	.0001*	.0001*
LDL-c (mg/dl)	r	0.8	0.9	0.9	0.9
	P-value	.0001*	.0001*	.0001*	.0001*

- P value < 0.05 is considered to be significant.

Discussion:

Diabetes is an important health problem since the incidence of diabetes is continuously increasing. Early diagnosis is important as type 2 diabetes begins long before we diagnose it, leading to a complicated course of the disease. In order to prevent delay in the diagnosis of type 2 diabetes, novel predictors and pathways for type 2 diabetes are mounting. Adipocytokines may play important roles in the pathogenesis of diabetes mellitus and insulin resistance (Gulcelik *et al.,* 2009).

The present study aimed to investigate the role of both adipokines; vaspin and chemerin in the pathogenesis of type 2 diabetes accompanied with obesity and the relationship between the two adipokines in the studied groups.

The current study showed that the mean serum level of vaspin in type 2 diabetic patients was significantly higher than that of the control group. Also, vaspin was significantly higher in obese type 2 diabetic patients than that of non obese type 2 diabetic patients.

These results came in agreement with a study done by El-Mesallamy *et al.* (2011) that revealed significantly elevated levels of vaspin in non obese and obese type 2 diabetic patients compared with control subjects.

In accordance to these results Youn *et al.* (2008) concluded that elevated vaspin serum concentrations are associated with obesity and impaired insulin sensitivity. Whereas type 2 diabetes seems to abrogate the correlation between increased circulating vaspin, higher body weight, and decreased insulin sensitivity.

Since vaspin is recognized as an insulin sensitizing adipokine, theoretically, we would expect a positive association between serum vaspin levels and HOMA. In our study, in type 2 diabetic patients, vaspin was significantly positively correlated with BMI, HOMA, TG, T- cholesterol, LDL-cholesterol and negatively correlated with HDL-cholesterol .

The administration of recombinant vaspin improved insulin sensitivity and glucose tolerance and reversed the expression of genes that may promote insulin resistance in diet-induced obese mice (Hida *et al.,* 2005). Therefore, reasonable speculation suggests that the production of vaspin may antagonize the action of unknown proteases that impair the action of insulin (Li *et al.,* 2008).

The present study showed that the mean serum level of chemerin in type 2 diabetic patients was significantly higher than that of the control group. Also, it was significantly higher in obese type 2 diabetic patients than that of non obese type 2 diabetic patients. In addition, chemerin, was significantly positively correlated with BMI, HOMA, TG, T- cholesterol, LDL-cholesterol and negatively correlated with HDL-cholesterol.

In accordance to these results Wang *et al.* (2009) concluded that serum chemerin levels were much higher in obese type 2 diabetic subjects than type 2 diabetic subjects with normal weight. Also, revealed that serum chemerin was correlated with insulin level, body fat disposition and lipid metabolism which suggesting that it may play a role in the pathophysiology of obesity and metabolic syndrome.

Additionally, Weiqert *et al.* (2010) revealed that circulating chemerin was similar in type 2 diabetes and obese individuals but was significantly elevated in both cohorts compared to normal weight individuals.

In contrast to our study Bozaoglu *et al.* (2007) concluded that plasma chemerin levels were not significantly different between subjects with type 2 diabetes and normal controls. However, in normal glucose tolerant subjects, plasma chemerin levels were significantly associated with body mass index and circulating triglycerides.

Although vaspin and chemerin levels seem to be associated with insulin resistance and glycemic status in our study, their validity as markers is questionable at present. Further investigations are needed to understand the regulation of vaspin and chemerin also, their role in the development of diabetes.

In conclusion, serum vaspin and chemerin levels are associated with insulin resistance in type 2 diabetic patients and positively correlated with HbA1c. Serum vaspin and chemerin levels are elevated in obese and non obese type 2 diabetic patients compared with controls.

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