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Chemerin Relationship with Glucose and Lipid Metabolism in Clinically Asymptomatic Patients

Ilze Skuja[1,2]*, Inga Stukena[1,2] and Aivars Lejnieks[1,2]

[1]Department of Internal Medicine, Riga Stradins University, Latvia
[2]Riga East University Hospital, Latvia

Abstract

Study background: Chemerin is one of the recently discovered adipocytokines, a chemoattractant protein consisting of 131 to 137 amino acids expressing mostly in adipocytes. Chemerin affects adipogenesis and glucose homeostasis, have a direct or indirect impact on the inflammatory response. International scientific literature studies the relationship between chemerin and diabetes, obesity and metabolic syndrome markers.

Methods: The study included 159 clinically asymptomatic patients (age 30-45) with an average age of 37.3 ± 4.0 years, in total 79 women.

The following anthropometric measurements and biochemical parameters were measured: weight, height, BMI and fasting glucose, insulin, glycated hemoglobin, total cholesterol with its fractions, liver enzymes and immunological marker-serum chemerin.

All patients underwent a CT examination of the liver with multilayer spiral computer tomograph. The density of the liver and spleen, liver index and liver/spleen index was determined.

Results: Increased BMI correlates with an increased chemerin level, HOMA-IR value and TG level. A chemerin level raise also elevates the ALT and GGT blood levels, as well as the mean liver density among all three groups. The liver index and the liver/spleen index is greater in the normal weight group compared with the overweight and obese groups.

HOMA-IR has a medium close-correlation with the chemerin level in the total group and overweight group. CT-derived liver indicators (mean liver density, liver index, liver/ spleen index) have a statistically significant but weak correlation with chemerin level both in the total group and overweight group.

Conclusion: Chemerin is adipokine, which has a significant, but not yet unambiguous role in the metabolic process. Chemerin correlation with almost all MS criteria show that even asymptomatic patients should pay attention even if only one MS criterion is increased, focusing on checking other criteria.

Keywords: Chemerin; Obesity; Metabolic syndrome; Insulin resistance; Liver density

Introduction

Chemerin

Chemerin is one of the recently discovered adipocytokines, a chemoattractant protein consisting of 131 to 137 amino acids expressing mostly in adipocytes [1]. Its effects are mediated by chemokine like receptor-1 known under the abbreviation CMKLR1, also known as ChemR23 [1,2]. CMKLR1 depends on chemerin secretion and differentiation [3,4]. Chemerin is synthesized only by adipocytes, whereas its receptor-CMKLR1-is synthesized by adipocytes, myeloid, plasmacytoid and dendritic line cells involved in innate and acquired immunity reactions [1,4]. Chemerin is secreted in an inactive form as prochemerin. Inflammatory and coagulation serine proteases cause cleavage of the C-terminus hence prochemerin becomes active and acts as a CMKLR1 ligand [2,5,6]. Approximately 106 differentiated adipocyte cells may secrete 15 ng/ml of chemerin within 48 hours [3]. This process slightly, but definitely is increased by the tumor-necrosis factor α (TNFα) and up to 80% is inhibited by the peroxisome proliferator-activated receptor activation [3]. One study has shown that adipocytes in obese patients with type 2 diabetes mellitus express chemerin significantly more than adipocytes of normal weighted patients without carbohydrate metabolism disorders (21 and 8 ng/ml from 107 cells, respectively) [3,4]. Chemerin was not found in skeletal muscle cells and macrophages, which were isolated from adipocytes [3]. Chemerin affects adipogenesis and glucose homeostasis [3,5-7]. In addition to the metabolic effects, scientific literature also describes reaction chains, which have a direct or indirect impact on the inflammatory response [1]. For example, chemerin stimulates macrophage adhesion to fibronectin, adhesion molecules ICAM-1 (intercellular adhesion molecule-1) and VCAM-1 (vascular cell adhesion molecule-1) [1]. These effects are mediated through CMKLR1 assistance, which is located on the macrophage membrane [1].

International scientific literature studies the relationship between chemerin and diabetes markers (HOMA - IR, HbA1c, fasting and general plasma glucose level), obesity markers (BMI, abdominal circumference, adiponectin, leptin) and metabolic syndrome markers (systolic blood pressure, diastolic blood pressure, low-density lipoprotein) [6]. In a number of scientific studies that researched the relationship of C-reactive protein (CRP) and chemerin level, a close correlation was found [8-11].

Chemerin and metabolic syndrome

Obesity is a worldwide problem causing versatile health issues

***Corresponding author:** Ilze Skuja, Riga Stradins University, Department of Internal Medicine, Latvia, Tel: 67847104; E-mail: skujailzedr@gmail.com

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such as insulin resistance, hyperglycemia, dyslipidemia, hypertension, and central obesity, as well as other signs and symptoms of metabolic syndrome (MS) [3,6,12].

Insulin resistance is one of the most characteristic manifestations of obesity. It causes MS development and visceral fat cell mass increase [3]. International literature describes that adipocytes act as a large secretory and endocrine organ, which secretes a variety of bioactive proteins that regulate the energetic metabolism and insulin sensitivity [3,13]. Three main functions of adipocytes are known in adults - energy accumulation, triglyceride hydrolysis and synthesis of adipokines [14]. Between adipocytes there are also stromal cells such as macrophages, monocytes and fibroblasts, which participate in the synthesis of adipokines, too [14].

Visceral fat has a role of consequence in the development of liver disease due to biologically active substances that affect the liver through the portal vein [15]. Studies have shown that adipokines are synthesized both by visceral and subcutaneous adipocytes [14]. Subcutaneous adipocytes synthesize adiponectin, leptin, retinol linking protein 4 and acylation stimulating protein [15]. The visceral adipocytes synthesize TNFα, IL6, IL8, visfatin, adipsin, plasminogen activator inhibitor 1, angiotensinogen and resistin [16].

The increase of adipocytes especially visceral ones is considered as one of the risk factors of type 2 diabetes mellitus [3,5,17,18]. Adipocytes of obese people have increased metabolic and endocrine activity; they secrete enhanced amount of inflammatory adipokines, such as tumor necrosis factor α (TNF α), interleukin (IL) 6, angiotensinogen, chemerin and resistin [3].

Chemerin and insulin resistance

International literature describes chemerin production's association with insulin resistance at lipogenesis level and adipocyte insulin-promoting antilipolysis [3]. Chemerin influences the activation of p38 mitogen protein, nuclear factor B and extracellular signal regulating protein kinase 1/2 [3].

Insulin resistance is characterized by a decrease of tissue receptor sensitivity to endogenous insulin, which is developed at a normal or even elevated quantity [13]. The cause of this phenomenon until now is unknown, however, the influence of hereditary factors has been proved [16]. Insulin resistance formation mechanism is promoted by TNFα, which phosphorylates the type 1 insulin receptor (IRS-1) decreasing its susceptibility to insulin and glucose transport in cells [19,20]. Many studies have reported that patients with insulin resistance have an increased amount of serum TNFα [21]. Adipocytes are considered to be the production sources of elevated TNFα, also possibly Kupffer cell activation due to bacterial antigens [21]. Recently the role of other adipocyte-synthesized mediators has been studied such as resistin, adiponectin, leptin and others [20]. Due to hyperinsulinemia, the b-oxidation of free fatty acids in the liver is diminished and very low-density lipoprotein growth in volume occurs, which contributes to the fatty liver dystrophy [19].

Studies have shown that the level of chemerin in diabetic patients rises more than in healthy people [3].

Non-alcoholic fatty liver disease (NAFLD)

Non-alcoholic fatty liver disease (NAFLD) is a widespread disease. It is one of the manifestations of the metabolic syndrome (MS) and is related to abdominal and visceral obesity, type 2 diabetes mellitus, dyslipidemia, hypertension, early atherosclerosis, coronary heart disease, polycystic ovarian syndrome and other diseases [20,21]. One of the main pathogenesis mechanisms of NAFLD is insulin resistance [20]. NAFLD is observed in 20-30% of the general population [21]. Children and young people account for 3-10%, rising up to 38-53% among children with obesity [15].

NAFLD can occur in two forms: fatty liver dystrophy and non-alcoholic steatohepatitis (NASH) [21]. The genes responsible for heredity of insulin resistance and NAFLD can be divided into three groups: those that regulate fatty acid oxidation, oxidation balance and expression of proinflammatory cytokine (TNFα) [19].

Ectopic fat such as visceral fat or omentum, epicardial and mediastinal fat play a significant role in promoting obesity [15]. Proper functioning of the fat tissue of those localizations is impaired and an inflammation develops, which also contributes to obesity [14,22]. Visceral fat is also very important in the development of liver disease, since the secreted substances affect the liver through the portal vein [15]. Studies have shown adipokine synthesis differences whether from visceral or subcutaneous adipocytes [14]. Namely, subcutaneous adipocytes synthesize adiponectin, leptin, retinol-binding protein 4 and acylation stimulating protein [15]. Visceral adipocytes synthesize TNFα, IL6, IL8, visfatin, adipsin, plasminogen activator inhibitor 1, angiotensinogen and resistin [15].

Aim

To evaluate chemerin level in relation with the basic criteria of metabolic syndrome (obesity, lipid and carbohydrate metabolism parameters) and NAFLD existence in clinically asymptomatic patients.

Material and Methods

Clinically asymptomatic patients from one GP's practice were invited to participate in the study. All patients received full explanation of the study and its objectives. For a patient to be included in the study, there were the following inclusion criteria:

1) Women and men aged 30-45 years,

2) Patient considers himself/herself healthy and does not use medication for chronic disease,

3) Patient has no signs of acute illness,

4) During examination there are no signs of a chronic disease,

5) Patient agrees to participate in the study and has signed the informed consent form.

Patients were excluded from the study if:

1) Patient had an acute illness or signs and symptoms of an acute illness in the past two months,

2) During the examination, an elevated systolic and/or diastolic blood pressure was detected for which medical treatment should be considered,

3) Patient had a chronic disease (chronic renal insufficiency with GFR<60 ml/min, thyroid hormone changes, chronic inflammatory systemic, joint and skin diseases),

4) Patient had had an oncologic disease in the last 5 years.

Riga Stradins University Ethics Commission approved the study and the study was in line with the Declaration of Helsinki.

The study included 159 patients with an average age of 37.3 ± 4.0 years, in total 79 women (mean age 37.2 ± 4.1 years) and 80 men (mean age 37.5 ± 4.0 years). The age of patients between sexes had

no statistically significant difference (p=0.983). Patients were divided into groups according to BMI. Depending on the BMI patients were divided into 3 groups: patients with normal weight (BMI 18.5 to 24.9 kg/m^2), overweight patients (BMI 25.0 to 29.9 kg/m^2) and patients with obesity (BMI ≥ 300 kg/m^2). The normal weight group included 53 patients with a mean age of 37.2 ± 3.7 years; the overweight group included 55 patients with a mean age of 37.9 ± 4.3 years; and the obesity group included 51 patients with a mean age of 36.8 ± 4.1 years. The age of patients between the groups did not have a statistically significant difference (p=0.790).

For each patient, the following anthropometric measurements were made: body weight and height (using certificated standardized methods); BMI was calculated using the formula: BMI = weight (kg)/height (m^2). Blood pressure was measured according to the recommendations of the European Society of Hypertension.

Blood tests were taken on an empty stomach after 12 hours of fasting at Ltd. "E. Gulbja Laboratorija". The following biochemical parameters were defined: glucose, insulin, total cholesterol (TC), HDL, TG, ALT, AST, GGT and glycated hemoglobin (HbA_{1c}) level in blood serum. LDL level was calculated using the formula Friedewald, also non-HDL (Non-HDL = TH (mmol/l)-HDL (mmol/l)) and insulin resistance (IR) index HOMA-IR (Homeostasis Model Assessment) (HOMA-IR=fasting glucose (mmol/l) × fasting insulin/22.5) were calculated. In addition, the patients' immunological marker-serum chemerin was defined using the ELISA immunological method (enzyme-linked immunosorbent assay) according to the manufacturer's instructions. Colorimetric reactions were read with the analyzer Infinite M200 (catalog No. EZHCMRN-57K).

All patients underwent a CT examination of the liver with multilayer spiral computer tomograph Somatom Definition 40. The density of the liver and spleen was determined using radiology adopted units-HU (Housfield Units). Six areas of interest were identified in 100 mm^2 field-4 in the right liver lobe, 2 in the left liver lobe and 3 areas in the spleen, where density was measured. The mean liver density was determined by calculating the average of the six chosen liver area measurements; liver index was determined subtracting the spleen mean density from the mean liver density, but the liver/spleen index was calculated as the ratio between the mean liver and spleen density.

The obtained data was analyzed using the SPSS 20.0 (Statistical Package for the Social Sciences) statistical program. The findings were checked for conformity with normal distribution using the Kolmogorov-Smirnov test. The parameters were analyzed and compared between groups using two independent sample t-test, if the data corresponded to a normal distribution and the Mann-Whitney test, if data did not correspond to a normal distribution. Correlation analysis was performed using Spearman's rank correlation coefficient. Data were considered to be statistically reliable by a significance level of p<0.05.

Results

Table 1 shows chemerin, as well as glucose and lipid metabolism biochemical marker values of patients stratified by BMI - normal weight, overweight and obese patients. As the data shows, increased BMI correlates with an increased chemerin level, HOMA-IR value and TG level. By contrast, glucose, HbA_{1c}, HDL and Non-HDL level is different between normal weight and overweight patients as well as normal weight and obese patients, however, these figures do not have a statistically significant difference between overweight and obese populations. Total cholesterol and LDL had no statistically meaningful difference between the three groups.

Data evaluated in Table 2 shows that a chemerin level raise also elevates the ALT and GGT blood levels, as well as the mean liver density among all three groups. Relatively statistically significant increase in both systolic and diastolic blood pressure in patients with normal blood pressure is observed. The liver index and the liver/spleen index is greater in the normal weight group compared with the overweight and obese groups, but no statistically significant difference between the overweight and obese group was found. The AST level of statistically significant difference was observed only between normal weight and obese patients.

Marker	Normal weight (N)*	Overweight (OW)*	Obesity (O)*	p**		
				Group N/OW	Group N/O	Group OW/O
Chemerin (ng/ml)	35.71 [29.72; 46.28]	41.79 [33.64; 54.35]	46.99 [42.69; 56.03]	0.036	<0.001	<0.001
Glucose (mmol/l)	4.80 [4.50; 5.09]	5.18 [4.80; 5.41]	5.30 [4.90; 5.70]	0.001	0.181	<0.001
HOMA-IR (mmol/l)	0.99 [0.53; 1.48]	1.84 [0.96; 2.85]	3.15 [2.22; 4.08]	<0.001	<0.001	<0.001
HbA_{1c}	5.50 [5.25; 5.60]	5.50 [2.30; 5.75]	5.6 [5.45; 5.95]	0.013	0.119	0.008
TH (mmol/l)	5.16 [4.68; 5.90]	5.54 [4.77; 6.42]	5.51 [4.82; 6.09]	0.246	0.702	0.156
HDL (mmol/l)	1.64 [1.46; 1.92]	1.43 [1.23; 1.71]	1.30 [1.15; 1.69]	0.003	0.156	<0.001
Non-HDL (mmol/l)	3.37 [2.86; 4.05]	4.01 [3.26; 4.84]	4.06 [3.43; 4.73]	0.013	0.672	0.002
LDL (mmol/l)	2.97 [2.50; 3.48]	3.45 [2.83; 4.14]	3.25 [2.73; 3.90]	0.057	0.456	0.166
TG (mmol/l)	0.81 [0.62; 1.06]	1.14 [0.85; 1.78]	1.45 [1.04; 2.12]	<0.001	019	<0.001

* If the data did not meet the normal distribution, average values were expressed as a median [I; III quartile].
** The significance level (p) of the difference between the two groups was calculated using independent sample t-test (normal distribution) or the Mann-Whitney test (if the data did not meet the normal distribution).

Table 1: Value of chemerin, glucose and lipid metabolism biochemical markers depending on the BMI and the significance level (p) between the groups.

Test	Normal weight (N)*	Overweight (OW)*	Obesity (O)*	p**		
				Group N/OW	Group N/O	Group OW/O
Chemerin (ng/ml)	35.71 [29.72; 46.28]	41.79 [33.64; 54.35]	46.99 [42.69; 56.03]	0.014	<0.001	0.030
SBP (mmol/l)	115 [105; 120]	120 [115; 125]	125 [115; 135]	0.001	0.020	<0.001
DBP (mmol/l)	70 [65; 80]	75 [70; 80]	80 [75; 80]	0.001	0.010	<0.001
ALT (mmol/l)	20.00 [17.00; 26.00]	22.00 [17.00; 37.00]	32.00 [24.00; 48.00]	0.032	0.013	<0.001
AST (mmol/l)	24.00 [21.00; 26.00]	24.00 [21.00; 29.00]	28.00 [23.00; 32.00]	0.208	0.098	0.003
GGT (mmol/l)	14.00 [10.00; 18.5]	19.00 [12.00; 36.00]	25.00 [17.50; 45.00]	0.005	0.003	<0.001
AVD (HU)	60.5 [58.00; 63.00]	58.00 [53.00; 61.00]	55.00 [49.75; 59.25]	0.002	0.004	<0.001
Liver index	11.00 [7.00; 14.00]	10.00 [5.00; 12.50]	8.50 [2.75; 12.00]	0.036	0.085	0.008
Liver/spleen index	1.22 [1.12; 1.27]	1.19 [1.08; 1.17]	1.17 [1.24; 1.18]	0.048	0.083	0.034

* If the data did not meet the normal distribution, average values were expressed as a median [I; III quartile].
** The significance level (p) of the difference between the two groups was calculated using an independent sample t-test (normal distribution) or the Mann-Whitney test (if the data did not meet the normal distribution).

Table 2: Chemerin, arterial blood pressure and liver enzyme average values depending on the BMI and the significance level (p) between the groups.

Group of patients		Parameter*	BMI	TH	HDL	Non-HDL	LDL	TG
Total		SCC	0.363	0.42	0.133	-0.433	0.531	0.290
		p	<0.001	<0.001	0.111	<0.001	0.001	<0.001
KMI	**Normal weight**	SCC	0.169	-0.099	0.017	-0.093	-0.087	0.170
		p	0.227	0.483	0.903	0.507	0.537	0.222
	Overweight	SCC	0.063	0.091	-0.199	0.084	0.012	0.178
		p	0.650	0.509	0.145	0.542	0.931	0.193
	Obesity	SCC	0.022	0.053	0.147	0.027	0.559	0.243
		p	0.878	0.118	0.710	0.304	0.850	0.086

* SCC -Spearman correlation coefficient; p - significance level.

Table 3: Correlation of chemerin level and lipid parameters in the total and separate patient groups depending on body mass index (BMI).

Group of patients		Parameter*	BMI	Glucose	HOMA-IR	HbA_{1c}	SBP	DBP
Total		SKK	0.363	0.128	0.460	0.214	0.531	0.290
		p	<0.001	0.159	<0.001	0.012	0.001	<0.001
KMI	**Normal weight**	SKK	0.169	0.053	0.268	0.193	0.053	0.239
		p	0.227	0.707	0.049	0.194	0.707	0.085
	Overweight	SKK	0.063	0.047	0.402	-0.043	0.077	0.008
		p	0.650	0.733	0.002	0.778	0.576	0.593
	Obesity	SKK	0.022	-0.085	0.211	0.233	0.063	0.031
		p	0.878	0.551	0.138	0.115	0.653	0.829

* SCC -Spearman correlation coefficient; p - significance level.

Table 4: Chemerin level correlation with glucose metabolism parameters and blood pressure in the total group and separate patient groups depending on body mass index (BMI).

Table 3 discusses chemerin level correlation with the lipid parameters and shows that the total group has a medium close correlation with BMI, TH, Non-HDL and LDL, but a weak correlation with TG. However, for patients of the separate groups this correlation was not statistically significant.

Glucose, HOMA-IR and HbA_{1c} correlation with chemerin is shown in Table 4. The acquired data shows that glucose level does not correlate with chemerin level neither in total nor individual BMI groups. HOMA-IR has a medium close-correlation with the chemerin level in the total group and overweight group, a weak correlation between the normal weight group, but no statistically significant correlation with the obesity group. Table 4 also shows that SBP, DBP and BMI correlate with the chemerin level in the total group, but by analyzing groups separately by the BMI, this correlation is not statistically significant.

Liver enzyme test correlations with chemerin are shown in Table 5. The data shows that both ALT and GGT have a statistically significant but weak correlation with chemerin level in the total group; when comparing the data in groups by BMI a statistically significant correlation between these indicators is no longer visible. AST has no statistically significant correlation with chemerin level. CT-derived liver indicators show that all three indicators (the mean liver density, liver index, liver/spleen index) have a statistically significant but weak correlation with chemerin level both in the total group and overweight group. In the normal weight and obesity group these figures did not correlate with the chemerin level.

Group of patients		Parameter*	BMI	AST	ALT	GGT	Mean liver density	Liver index	Liver/ spleen index
Total		SCC	0.363	0.141	0.253	0.294	-0.250	-0.102	-0.109
		p	<0.001	0.076	0.001	<0.001	0.002	0.021	0.017
BMBI	Normal weight	SCC	0.169	0.120	-0.027	0.156	-0.056	0.054	0.038
		p	0.227	0.929	0.849	0.264	0.697	0.712	0.789
	Overweight	SCC	0.063	0.137	0.214	0.171	-0.268	-0.234	-0.279
		p	0.650	0.318	0.117	0.213	<0.001	0.048	0.039
	Obesity	SCC	0.022	0.026	0.190	0.209	-0.069	0.054	0.005
		p	0.878	0.856	0.182	0.141	0.635	0.711	0.973
* SCC -Spearman correlation coefficient; p - significance level.									

Table 5: Chemerin level correlation with liver enzyme tests and CT acquired data in the total group and in patient groups depending on body mass index (BMI).

Discussion

Various scientific studies have found conflicting data on chemerin association with MS components. Several studies have found a strong link between triglycerides (TG), total cholesterol (TC) and chemerin blood level [4,6,8-11,23-25]. Comparing the relationship between chemerin, the TG and TC, TG direct relationship is closer [6]. In our study the chemerin level in the overall group - just like in aforementioned research - had a close correlation with TC and TG, although the closest correlation was found with TC instead of TG. Individual groups by BMI did not prove this correlation due to the small number of patients in a group. Some studies did not prove HDL and LDL correlation with the chemerin level [4,6,8-11,23-25], however our study showed a chemerin level correlation with LDL and non-HDL, but the same was not observed for HDL. These contradictions also confirmed the discussion in the scientific literature of proving a chemerin relationship with dyslipidemia.

The scientific literature describes chemerin level elevation as increasing the human body mass index (BMI) above 25 kg/m^2, compared to people with a BMI below 25 kg/m^2 [3,6]. Also, our study analyzing the average chemerin level by BMI groups proved a statistically significant increase in overweight and obese patient groups when compared to normal weight. Abdomen and hip circumference, BMI and fat cell volume have a close correlation with the secretion of chemerin [3]. Our study's overall group chemerin correlation with BMI was medium close. A study of obese patients undergoing bariatric gastric surgery showed that chemerin level reduction was due to weight reduction and improvement in metabolic parameters [6,26,27].

However, scientific literature describes other studies having found a strong correlation between MS components, such as BMI, triglycerides (TG), low density lipoprotein cholesterol (LDL-C), arterial hypertension and chemerin level [4,6,23,26]. This data has also been proven in our study results. Out data shows that even patients with normal blood pressure have a medium strong (for SBP) and weak (for DBP) correlation with their chemerin level. SBP, DBP and chemerin level elevates group-by-group for patients with normal weight, overweight and obesity. These results confirm what some studies have shown – by measuring chemerin level in patients with diabetes mellitus a significant difference was not found, while in healthy individuals it was positively correlated with both BMI, lipid levels, insulin, adiponectin and arterial hypertension [3,4,11].

Scientific literature demonstrates that insulin resistance parameters such as HOMA-IR are closely correlated with the chemerin blood level [4,27]. It was also confirmed by our study results since HOMA-IR index was the only parameter of glucose metabolism that was statistically significantly different between all three BMI groups just like chemerin. Similarly, the only HOMA- IR correlation with chemerin was shown in all evaluated groups. Our study also demonstrates that pathogenically the most important chemerin role is in insulin resistance development. In the obese patient group it was different between normal weight patients and patients with increased weight, which coincides with the chemerin level difference between completely healthy and sick individuals described in scientific research.

Worldwide a lot of research has been done about the relationship between non-alcoholic fatty liver disease and chemerin, however conflicting data are found. Some studies have shown that chemerin level is not elevated in patients with non-alcoholic fatty liver disease compared to healthy subjects [24], while other studies show otherwise - chemerin levels are higher in patients with non-alcoholic fatty liver disease [28-30]. In our study, both the total group and overweight group showed an liver attenuation, liver index and liver/spleen index decrease with a chemerin level increase.

Non-alcoholic fatty liver disease cases are often characterized with elevated liver serum transaminases, but the international available studies usually do not find an ALT relation with the blood chemerin level [4,6,8-10,24,31,32]. Only some studies have demonstrated such relevance [28]. Also our study shows a weak correlation between chemerin level, ALT and GGT, which may be associated with group distribution and a larger number of patients, with NAFLD in the obese patient group compared with the normal weight patients group.

Conclusions

Chemerin is adipokine, which has a significant, but not yet unambiguous role in the metabolic process. It can be used as an early detection for glucose and lipid metabolism disorders that might earlier reveal diabetes mellitus, atherosclerosis, CVD and NAFLD patients with whom classic disease symptoms have not yet manifested.

Chemerin correlation with almost all MS criteria show that even asymptomatic patients should pay attention even if only one MS criterion is increased, focusing on checking other criteria.

Chemerin level changes show that for clinically asymptomatic patients with obesity, it would be desirable not only to assess the blood glucose level but also the feasibility of insulin resistance, while glycated hemoglobin testing must be evaluated whether it is sufficiently justified and useful for overweight and obese patients.

In order to assess the risk of CVD and metabolic changes in obese patients in addition to glucose, TG and HDL level assessment is appropriate to assess insulin resistance, liver transaminases, as well as changes in the above mentioned indicators chemerin blood level and liver density.

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