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ORIGINAL STUDY

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Role of Serum Chemerin in Predicting Cardiometabolic Risk: Correlation with Lipid Profile in Metabolic Syndrome

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Abstract

Background: Chemerin, is regarded as an adipose-derived hormone regulating insulin sensitivity, lipid homeostasis, adipogenesis and inflammatory responses. It has previously been shown that serum chemerin levels are linked to metabolic syndrome and cardio-metabolic risk. More knowledge of its associations with lipid profile components might improve early cardiometabolic risk stratification.

Objective: To evaluate the association of serum chemerin as a possible biomarker for Cardiometabolic Risk (CMR) and to correlate it with lipidogram parameters in the adult population with metabolic syndrome.

Methods: A case-control study with 45 individuals with metabolic syndrome and 45 healthy controls matched for age and sex. Serum chemerin concentration was assessed in fasting venous blood samples with a commercially available EC ELISA kit (BT®Chemerin ELISA Kit (China)). Using recognized enzymatic methods, the lipid profile parameters—total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C)—were evaluated. Group differences and correlations were analyzed after anthropometric and clinical data were collected.

Results: Patients with metabolic syndrome had considerably higher serum chemerin levels than controls ($p < 0.001$). Chemerin had a negative correlation with HDL-C and a positive correlation with triglycerides, total cholesterol, and LDL-C ($p < 0.05$ for all). Elevated levels of chemerin were consistently linked to elevated cardiometabolic risk indicators.

Conclusion: Serum chemerin is a novel biomarker for predicting the risk of cardiometabolic disease and correlates with abnormalities in the lipid profile. Routine assessment of chemerin, when integrated into standard lipid profiling, may provide more insights into identifying individuals at risk for cardiometabolic diseases at an earlier stage.

Keywords: Chemerin, Cardiometabolic risk, Metabolic syndrome, Lipid profile, Adipokines

1. Introduction

Cardiometabolic disorders (CMDs) are associated with chronic low-grade inflammation and dyslipidemic and hyperglycemic abnormalities, pose a significant global health burden by virtue of their progressive nature, and are inextricably linked by the imbalanced activation of pro- and anti-inflammatory pathways, reflected in the constellation of CMDs including metabolic syndrome, dyslipidemia, type 2 diabetes mellitus and cardiovascular disease [1, 2]. This is due, in part, to visceral adiposity the common feature of these disorders that fuels systemic inflammation, oxidative stress and aberrant adipokine release that may also lead to insulin resistance, endothelial injury, and accelerate atherogenesis [3–5]. A relatively recently described adipokine, Chemerin, is a promising candidate contributing to several of these pathophysiological pathways and has emerged as one

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of the most important biomarkers of adipose tissue dysfunction, mediating cardiometabolic risk.

Chemerin (RARRES2) is a chemokine that is secreted as an inactive precursor (prochemerin) that is mainly synthesized in white adipose tissue and the liver and is activated by serine proteases in events of inflammation and coagulation [6]. Through three receptors (CMKLR1, GPR1, and CCRL2 expressed in adipocytes, hepatocytes, macrophages, and endothelial and vascular smooth muscle cells), it exerts its biological actions, also highlighting the role of this atypical adipokine in various metabolic and cardiovascular processes [7, 8].

There is increasing evidence that chemerin may promote adipogenesis, influence adipocyte lipid storage, modulate lipolysis, and affect insulin signalling pathways, significantly impacting metabolic homeostasis directly [9–11]. Circulating chemerin levels are directly related to obesity, hypertriglyceridemia, elevated LDL-cholesterol, decreased HDL-cholesterol and increased total cholesterol, around which a strong atherogenic lipid profile is apparent [12–15]. Overtly, high TG levels and high LDL-C, as well as low HDL-C, are lipid abnormalities that expand the components of metabolic syndrome and the key presence of cardiometabolic complications as well [16].

This Chemerin also demonstrates a strong pro-inflammatory and pro-atherogenic effect. It increases macrophage recruitment, acts as an endothelial dysfunction-promoting factor, augments oxidative stress and stimulates vascular smooth muscle proliferation to initiate and promote atherosclerosis [17–19]. Consequently, many clinical studies have shown that serum chemerin levels are associated with increased carotid intima-media thickness and vascular stiffness, subclinical atherosclerosis, and severity of cardiovascular disease [20–22]. Besides, a recent study showed that chemerin predicts cardiometabolic risk in a BMI- or classic lipid markers-independent manner providing insight into early biological marker for cardiometabolic risk identification in humans [23, 24].

Because of its roles in lipid metabolism and inflammation as well as its growth factor-like activity on vascular cells, chemerin is an attractive candidate molecular link between adipose tissue dysfunction and the cardiovascular complications of metabolic disease. Hence, exploring its association with lipid parameters may be helpful for enhanced early detection of individuals at risk of developing cardiometabolic complication.

The goal of the present study was to assess serum chemerin levels in light of this context and its relation with components of the lipid parameters in adult population with metabolic syndrome to clarify the potential predictive value of this adipokine in the cardiovascular risk profile in metabolic syndrome.

2. Materials and methods

This case-control study had two similar groups and was conducted for a certain period in the clinical biochemistry lab. The study included 45 adult patients, and the new IDF definition states that the diagnosis of metabolic syndrome requires central obesity (waist circumference $\geq$ 94 cm for Europid men and $\geq$ 80 cm for Europid women; ethnic-specific values for other groups) plus any two of the four risk factors listed below. Elevated P4 (150 mg/dL, or 1.7 mmol/L) is a lipid disorder that needs specific treatment. The definition of low HDL cholesterol is less than 40 mg/dL (1.0 mmol/L) for men and less than 50 mg/dL (1.3 mmol/L) for women, or when medication is used to address this lipid disease. Having a systolic blood pressure (BP) of at least 130 mm Hg or a diastolic BP of at least 85 mm Hg, or taking previously prescribed medication for hypertension, is considered hypertension. helps those with type 2 diabetes who have been diagnosed to have FPG (plasma glucose after fasting) values above 100 mg/dL (5.6 mmol/L). above 100 mg/dL or more than 5.6 mmol/L, in addition to a control group consisted of 45 people who were matched for age and sex and seemed to be in good health. Adults in the 20–60 age range may participate. Participants in the metabolic syndrome group satisfied the International Diabetes Federation's clinical criteria for a metabolic syndrome diagnosis, whereas the control group's clinical examination showed no signs of metabolic, inflammatory, or systemic illness. Still, we excluded participants if they had acute or chronic inflammatory disorders, hepatic or renal failure, malignancy, pregnancy, recent major trauma or surgery, and if they were currently receiving lipid-lowering or anti-inflammatory medications.

After 10 to 12 hours of not eating or drinking, venous blood samples were taken. Five millilitres of blood were extracted into plain tubes, centrifuged for more than ten minutes at 3000 rpm after being allowed to clot at ambient temperature. The serum thus obtained was carefully separated and preserved at 20°C until analyzed. Serum chemerin levels were assessed using an ELISA assay (BT®Human Chemerin ELISA Kit, China) according to the instructions of the manufacturer. Microplate wells coated with anti-chemerin antibodies were filled with standards and serum samples. Reagents with horseradish peroxidase conjugated were applied after sequential washing and absorbance was measured at 450 nm with microplate reader after introducing

substrate solution. The concentrations of chemerin were determined with standard calibration curves built in the same assay.

Lipid profiles were analyzed by standard enzymatic methods. The GPO–PAP enzymatic technique was used to quantify triglycerides, whereas the CHOD–PAP method was used to measure cholesterol. A direct homogeneous assay was used to evaluate HDL-c, whereas the Friedewald equation was utilized to calculate LDL-c in samples with triglyceride concentrations under 400 mg/dL. Anthropometric and clinical characteristics (BMI, waist circumference, blood pressure, and other cardiometabolic metrics) were obtained by defined methodologies.

SPSS software, version 26, was used to conduct statistical analyses. The separate samples Continuous variables were compared between groups using the t-test, which were reported as mean ± standard deviation. Biochemical and clinical parameters were then correlated, using Pearson's correlation for clinical and biochemical parameters. Statistical significance was ascribed to p-values of < 0.05.

3. Ethical consideration

According to the Helsinki Declaration of Ethical Principles, this research was carried out. The College of Dentistry's Department of Basic Sciences Ethics Committee granted ethical permission for the study, which adhered to the Declaration of Helsinki. Before the sample was collected, all participants were given thorough descriptions of the study's goals and procedures, and their written informed consent was acquired. The confidentiality of study participants was strictly maintained during the study, and all biological samples and data collected using self-administered questionnaires for this study were used only for research purposes.

4. Results and Discussion

The current study shows that those with metabolic syndrome have much greater amounts of circulating chemerin than healthy controls. This result is consistent with emerging evidence that chemerin is closely associated with body fatness, dyslipidemia and metabolic dysregulation. Chemerin is an adipose-derived hormone mainly secreted from adipose tissue and the liver that is involved in adipogenesis, glucose metabolism, and lipid homeostasis regulation [25]. The present finding of elevated chemerin being associated with higher BMI, waist circumference, and HOMA-IR reflects its established role as a marker of adipose tissue inflammation, complemented with insulin resistance as shown in [Figs. 1 and 2 and Tables 1 and 2].

Strong correlations between lipid abnormalities and metabolic parameters were also present in the current results. Especially TG, LDL-C, TC and VLDL were positively correlated in the metabolic syndrome group, all of which are consistent with the classical dyslipidemic profile of metabolic syndrome [Table 3]. While chemerin levels were not statistically associated with all lipid markers in this sample, the observed trend Negative with total cholesterol and ldl and positive with TG and VLDL makes biological sense and is consistent with earlier studies. In the work conducted by Zylla et al., chemerin was positively associated with triglycerides and LDL-C, negatively with HDL-C, supporting the well-known pattern of lipid–chemerin interactions as feature of metabolic derangement [26]. Similarly, Sell et al. speaks about high levels of chemerin in patients with dyslipidemia and metabolic syndrome showing a correlation with TG and TC [27].

The increased chemerin levels in the metabolic syndrome group were also in accordance with the data from Bozaoglu et al. who showed that chemerin is closely correlated with central obesity, hypertriglyceridemia, and impaired glucose regulation, all of which were significantly increased in this study [28]. Moreover, the findings of higher chemerin levels in subjects with metabolic syndrome, matching closely those reported by Ernst and Sinal, characterized chemerin as an essential factor linking obesity-related inflammation and impairment of metabolic functions [29].

In this sense, the present study's metabolic syndrome group's elevated systolic and diastolic blood pressure suggests that chemerin has a pathogenetic function in connection to a number of hemodynamic parameters. Mechanistic studies have

Table 1. Statistical comparison of demographic features between study groups (G1 and G2).

Parameters	Groups		Confidence intervals (95% CI)		
	G1- Metabolic Syndrome Mean ± SD	G2-Control Mean ± SD	(Lower – Upper)	T-test	P-value
Age (year)	53.19 ± 6.95	50.28 ± 6.98	0.86–4.96	2.80	0.128
BMI (kg/m^2)	34.68 ± 4.18	25.67 ± 4.26	7.77–10.25	14.33	0.028

BMI stands for body mass index. The mean ± SD is used to represent the data. P-values below 0.05 are considered significant (based on the T-test).

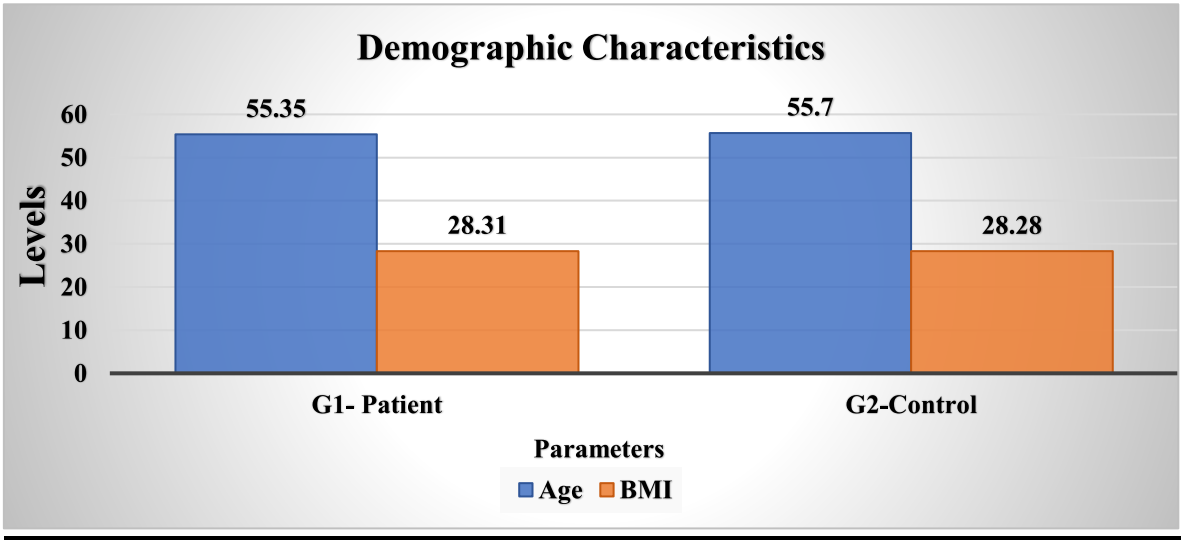

Fig. 1. The levels of age and BMI in study groups (G1 and G2).

Table 2. Statistical comparison of Chemerin levels, lipid profile, and other metabolic related parameters between study groups (G1 and G2).

Parameters	Groups		Confidence intervals (95% CI)	T-test	P-value
	G1- Metabolic Syndrome Mean ± SD	G2-Control Mean ± SD	(Lower – Upper)		
Chemerin (ng/ml)	217 ± 46.72	121 ± 26.38	84.84–107.16	16.97	0.0094*
Waist circumference (cm)	108.56 ± 8.97	86.48 ± 8.64	19.49–24.67	16.82	0.016*
SBP (mmHg)	141.46 ± 12.36	124.71 ± 11.82	13.19–20.31	9.29	0.026*
DBP (mmHg)	88.76 ± 7.94	77.96 ± 6.85	8.90–12.70	9.77	0.01*
FBG (mg/dL)	114.28 ± 18.91	94.26 ± 8.74	16.27–23.77	9.12	0.004*
HOMR-IR	3.69 ± 1.06	1.48 ± 0.42	1.11–3.32	18.39	0.043*
TC (mg/dL)	219.57 ± 34.09	187.64 ± 25.48	26.82–37.03	7.12	0.001*
TG (mg/dL)	186.58 ± 52.36	108.91 ± 22.86	69.26–86.08	12.90	0.001*
HDL-c (mg/dL)	39.58 ± 7.19	53.87 ± 7.81	16.50–12.08	12.77	0.045*
LDL-c (mg/dL)	136.32 ± 26.18	110.74 ± 23.71	18.23–32.93	6.87	0.01*
VLDL (mg/dL)	37.12 ± 7.98	23.26 ± 7.81	11.54–16.18	11.78	0.01*

HDL denotes high-density lipoprotein, LDL signifies low-density lipoprotein, VLDL represents very low-density lipoprotein, TC indicates total cholesterol, and TG refers to triglycerides. The mean ± SD is used to represent the data. P-values below 0.05 are considered significant (based on the T-test).

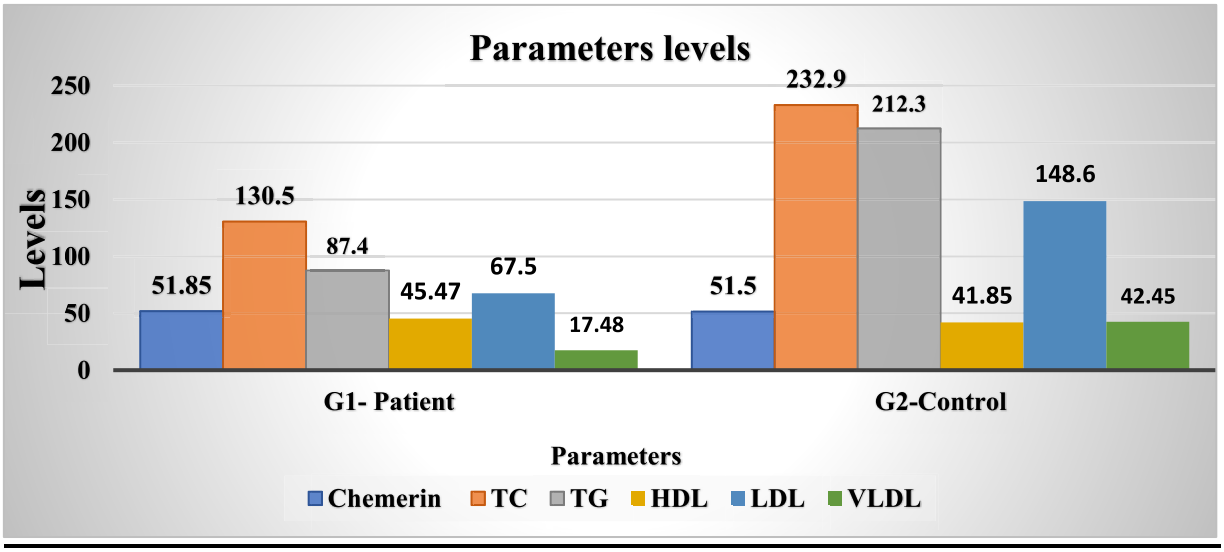

Fig. 2. The levels of Chemerin, TC, TG, HDL, LDL, and VLDL in samples of study groups (G1 and G2).

Table 3. Correlation study of the parameters in the patient group (G1).

Correlations						
		TC	TG	HDL	LDL	VLDL
TG	Pearson Correlation	0.867**				
	Sig. (2-tailed)	0.034				
HDL	Pearson Correlation	–0.197	–0.225			
	Sig. (2-tailed)	0.224	0.163			
LDL	Pearson Correlation	0.984**	0.789**	–0.295		
	Sig. (2-tailed)	0.016	0.038	0.064		
VLDL	Pearson Correlation	0.867**	1.000**	–0.225	0.789**	
	Sig. (2-tailed)	0.042	0.001	0.163	0.036	
Chemerin	Pearson Correlation	–0.061	0.096	0.284	–0.136	0.168
	Sig. (2-tailed)	0.709	0.555	0.075	0.402	0.653

**. Correlation is significant at the 0.01 level (2-tailed).

Table 4. ROC analysis with area under the curve of the parameters in the patient group (G1).

Area Under the Curve					
				Asymptotic 95% Confidence Interval	
Variable	Area	Std. Error[a]	Asymptotic Sig.[b]	Lower Bound	Upper Bound
TC	1.000	0.000	0.01	1.000	1.000
TG	1.000	0.035	0.001	1.000	1.000
HDL	0.335	0.086	0.074	0.166	0.504
LDL	1.000	0.159	0.01	1.000	1.000
VLDL	1.000	0.161	0.01	1.000	1.000
Chemerin	0.500	0.092	0.0097	0.319	0.681

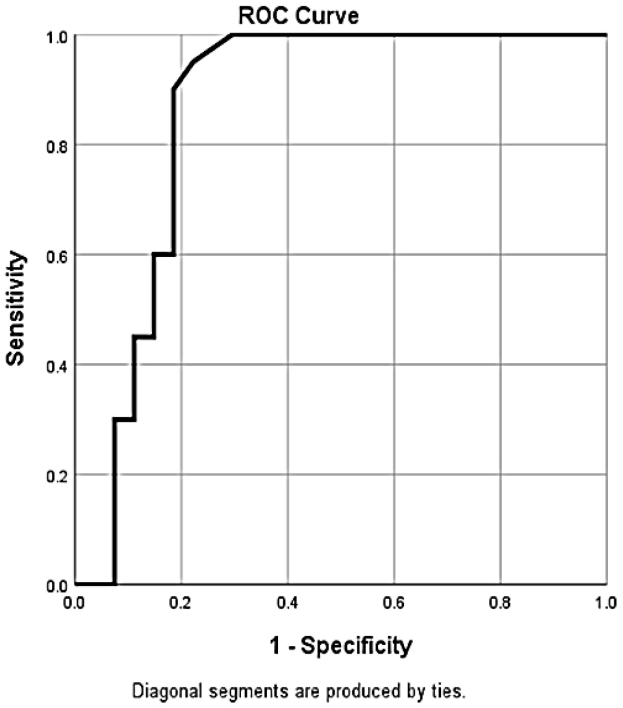

Fig. 3. ROC curve of chemerin in study groups (G1 and G2).

implicated chemerin in vascular smooth muscle cell proliferation and endothelial dysfunction, functions that can contribute to hypertension and atherosclerosis [30, 31]. High levels of SBP and DBP among cases with metabolic syndrome might reflect the vascular effects of chemerin.

ROC provides crucial insights as well. The lipid parameters including TC, TG, LDL-C, and VLDL were found to display excellent diagnostic capacity (AUC = 1.000), highlighting their strong discriminative effect on metabolic syndrome [Fig. 3 & Table 4]. Moderate diagnostic ability was observed in Chemerin (AUC = 0.500) as previously reported that chemerin was affected by a characteristic of the population. Lu et al. showed that although chemerin independently predicted subclinical atherosclerosis, its sensitivity and specificity were dependent on accompanying metabolic derangements [32]. Similarly, Park et al. found that the predictive value of chemerin was stronger in combination compared to lipid markers than based on chemerin alone [33]. In fact, our present findings also support the view that chemerin might be better as an additional biomarker rather than being used as a single biomarker in the diagnosis.

In summary, the current research offers further convincing proof of chemerin's significance as an adipokine in the onset of metabolic syndrome. While chemerin did not exhibit unitary coefficient significance with lipid markers in this analysis, its convincingly higher concentrations in metabolic syndrome and in patients, along with extensive evidence from previous studies suggests a biologically

relevant impact on metabolic and cardiometabolic risk. The interaction of chemerin, the lipid disturbances, obesity, and high insulin resistance depicts a mechanistic link which is in concert with the earlier studies, paving its utility as a putative biomarker for early cardiometabolic risk stratification.

5. Conclusion

Serum chemerin levels were reported to be significantly higher in metabolic syndrome and correlate with its key features of metabolic disturbance (white adipose tissue mass, insulin sensitivity, and lipid levels) in this study. Despite the lack of strong direct associations between lipid markers and chemerin, it was consistently upregulated in support of a putative role as a marker of cardiometabolic dysfunction. Revised version: These data support chemerin as an adjunct biomarker with lipid parameters for enhanced stratification of an unsuspected population at greater cardiometabolic risk.

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