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Original Article

Biochemistry Section

Serum Omentin-1 Levels in Patients with Type 2 Diabetes Mellitus and their Correlation with Lipid Profile: A Case-control Study

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ABSTRACT

Introduction: Type 2 Diabetes Mellitus (T2DM) is characterised by a variable degree of insulin resistance, leading to hyperglycemia. Serum omentin-1, an important adipocytokine, plays a role in maintaining tissue sensitivity to insulin and lipid metabolism. Therefore, circulating omentin-1 can serve as a biomarker for obesity, diabetes, and dyslipidemia.

Aim: The aim of this study was to compare serum omentin-1 levels between type 2 diabetic cases and non diabetic controls, and to investigate the correlation of omentin-1 levels with the lipid profile.

Materials and Methods: A hospital-based case-control study was conducted at Government Medical College, Trivandrum, from December 2017 to November 2018. The study included 30 T2DM patients and 30 non diabetic controls. Serum omentin-1 levels, fasting blood sugar, glycated haemoglobin (HbA1C), and serum lipid profile were measured in all study subjects. Statistical analysis included Student's t-test and Pearson's correlation coefficient to assess the correlation between variables. Chi-square test was performed to analyse age and sex distribution among the study groups.

Results: The mean age of diabetic cases was 56.7±7.9 years, while the mean age of non diabetic controls was 52.9±5.5 years. Among the subjects, 43% of diabetics and 50% of non diabetics were male, while 57% of diabetics and 50% of non diabetics were female. The mean value of serum omentin-1 levels was significantly lower in the diabetic population (273.37±60.18 ng/mL) compared to the non diabetic controls (573.4±51.6 ng/mL) with a p-value of <0.001. Serum omentin-1 showed a significantly strong negative correlation with total cholesterol, low-density lipoproteins (LDL), and triglycerides, and a positive correlation with high-density lipoproteins (HDL).

Conclusion: In the present study, serum omentin-1 levels were significantly lower in the diabetic population, and their lipid profile exhibited abnormal values. This suggests that omentin promotes insulin sensitivity and affects lipid metabolism, thus preventing insulin resistance and dyslipidemia in T2DM patients. Therefore, omentin may play a role in reducing cardiovascular complications.

Keywords: Cardiovascular complications, Dyslipidaemia, Fasting blood sugar, Insulin resistance

INTRODUCTION

Diabetes Mellitus (DM) is one of the largest epidemics the world has faced, both in developed and developing nations. The International Diabetes Federation (IDF) estimated that in 2015 there were 415 million people with DM and that by 2040 the number will be 642 million [1]. China and India are the top two epicentres in the rapidly emerging T2DM global epidemic [2].

Early developmental factors (such as intrauterine exposures), genetic predisposition, an unhealthy diet, obesity, and a sedentary lifestyle are important etiological factors for T2DM [2]. The most common cause of insulin resistance in humans is obesity [3,4].

Due to its multiple effects on fat metabolism, T2DM results in the production of atherogenic dyslipidaemia characterised by elevated Very-Low-Density Lipoprotein (VLDL), elevated LDL, elevated Triacylglycerol (TAG), and decreased HDL cholesterol. This dyslipidaemia is attributed to insulin resistance [5].

Omentin-1 is a recently identified adipocytokine [a bioactive cytokine synthesised from Visceral Adipose Tissue (VAT)] consisting of 313 amino acids. It is expressed in visceral (omental and epicardial) fat, as well as mesothelial cells, vascular cells, airway goblet cells, small intestine, colon, ovary, and plasma [6]. Omentin-1 and Omentin-2 are the two isoforms found in human circulation, with omentin-1 being the predominant isoform [6]. The important role of serum omentin-1 is to maintain the sensitivity of tissues to insulin. Omentin-1 enhances insulin-stimulated glucose uptake via Akt activation in human adipocytes [6,7].

Omentin, through Adenosine Monophosphate (AMP)-activated protein kinase signaling, acts as an anti-inflammatory, antiatherosclerotic, and cardiovascular protective agent [6]. Whenever there is an excess of VAT, as in obesity, an imbalance occurs among the adipocytokines released from the adipose tissues. This imbalance is characterised by increased production of many adipocytokines, such as Tumour Necrosis Factor-α (TNF-α), interleukin-6, etc., and decreased production of adiponectin, omentin, and vaspin, which are beneficial in the context of obesity-linked complications and metabolic disorders, including dyslipidaemia [6]. Circulating omentin-1 values can be used as a biomarker for obesity, diabetes, dyslipidaemia, hypertension, atherosclerosis, ischaemic heart disease, inflammatory disease, and cancer, as these diseases are characterised by excess VAT [6]. Therefore, it would be useful to study hormones that promote and decrease the sensitivity of tissues to insulin.

Although there are studies suggesting that reduced omentin levels are the cause of insulin resistance [8,9], the novelty of this study is that it also highlights the important role of omentin-1 in lipid metabolism, thereby preventing dyslipidaemia and cardiovascular complications and related morbidity and mortality in T2DM patients. Thus, omentin may be considered a therapeutic target and marker for predicting cardiovascular complications in T2DM patients.

Hence, the present study was conducted to compare serum omentin-1 values in T2DM patients and non diabetic controls at Govt. Medical College, Trivandrum, and to evaluate the correlation between serum omentin-1 values and serum lipid profile in T2DM patients and non diabetic controls.

MATERIALS AND METHODS

This hospital-based case-control study was conducted at the Government Medical College, Trivandrum, Kerala, India, over a

one-year period from December 2017 to November 2018. The study obtained clearance from the Human Ethics Committee of Government Medical College, Thiruvananthapuram, on 24/11/2017 (IEC.NO.13/11/2017/MCT).

Inclusion criteria: The study included 30 patients with T2DM who were willing to give consent and had an age of onset of the disease ≥40 years. These patients were receiving treatment with oral hypoglycemic drugs. The selection of patients with T2DM was based on the standard criteria set by the World Health Organisation (WHO), which included fasting blood sugar values ≥126 mg/dL and Glycated hemoglobin levels/HbA1c ≥6.5% [10]. Additionally, the study involved 30 age and sex-matched non diabetic controls who were willing to give consent.

Exclusion criteria: Patients who were not willing to give consent, patients on insulin therapy, patients with systemic illness or acute infections, and patients with gestational diabetes mellitus (DM) were excluded from the study.

Sample size calculation: The sample size was determined using the formula for comparing means of two groups based on a previous study [9]. The calculated sample size was 30 diabetic cases and 30 non diabetic controls.

$$n=\frac{2\times(Z_{1-\alpha/2}+Z_{1-\beta})^2\times\sigma^2}{d^2}$$

$$\sigma=\frac{\sigma1+\sigma2}{2}$$

σ is the standard deviation

$Z_{1-\alpha/2}=1.96$

$Z_{1-\beta}=0.84$

d / difference between mean (mean of non diabetic controls is 27.4 and diabetic cases is 19.7 and d is 7.7) [9].

Study Procedure

Demographic data, such as age, gender, weight, height, and Body Mass Index (BMI), were collected from all the study subjects. Body weight was measured using a standard weighing machine in kilograms, and height was measured with a wall-mounted scale in centimeters. BMI was calculated as weight (in kg)/height (in meters)2, with a BMI of 18.5 to 24.9 considered normal [10].

Patients were advised to fast for 8 hours and 5 to 7 mL of blood samples were collected by antecubital venipuncture with strict aseptic precautions. Serum fasting omentin-1 levels (5-800 ng/mL) were assessed using commercially available human serum omentin-1 kits (KINESIS Dx USA). Fasting Blood Sugar (FBS) (reference range: 70-110 mg/dL) was determined using the Hexokinase method [10]. HbA1c (reference range: <6.5%) was determined with whole blood from a fully automated analyser using the immunoturbidometric method [10]. Serum total cholesterol (reference range: <200 mg/dL), triglyceride (reference range: <150 mg/dL), and HDL (reference range: >60 mg/dL) were measured using standard enzymatic techniques on a fully automated analyser [10]. LDL (reference range: <100 mg/dL) cholesterol was estimated using the Friedewald equation [10].

STATISTICAL ANALYSIS

The data were entered into a personal computer using the Microsoft Excel Worksheet computer package. For data analysis, the Statistical Package for Social Sciences (SPSS) version 22.0 for Windows was utilised. The data were found to be normally distributed. Statistical tests, such as Student's t-test and Pearson's correlation coefficient, were performed to determine the correlation between variables. The Chi-square test was conducted to examine the age and sex distribution among the study groups. A p-value of <0.05 was considered statistically significant.

RESULTS

A total of 60 patients were included in the study, comprising 32 female patients and 28 male patients. Among them, 30 were diabetic cases with a mean age of 56.7±7.9 years, and 30 were non diabetic controls with a mean age of 52.9±5.5 years.

Regarding age distribution, 8 (26.7%) diabetic cases and 12 (40%) non diabetic controls were in the age range of 40-50 years, while 8 (26.7%) diabetic cases and 3 (10%) non diabetic controls were over 60 years of age. [Table/Fig-1] provides a summary of the age distribution, mean age, and sex distribution.

Parameters	Diabetic (n=30) n (%)	Non diabetic (n=30) n (%)	Total n (%)	p-value
Age (in years)				
40-50	8 (26.7)	12 (40)	20 (33.3)	0.334
51-55	7 (23.3)	9 (30)	16 (26.7)	
56-60	7 (23.3)	6 (20)	13 (21.7)	
>60	8 (26.7)	3 (10)	11 (18.3)	
Mean age	56.7±7.9	52.9±5.5		
Gender distribution	n (%)	n (%)	n (%)	0.605
Male	13 (43.3)	15 (50)	28 (46.7)	
Female	17 (56.7)	15 (50)	32 (53.3)	
Total	30 (100)	30 (100)	60 (100)	

[Table/Fig-1]: Age and sex distribution among the study group.
Chi-square test.
[1]χ^2 (chi-square)=3.40 df (degree of freedom)=3
[2]χ^2 (chi-square)=0.268 df (degree of freedom)=3

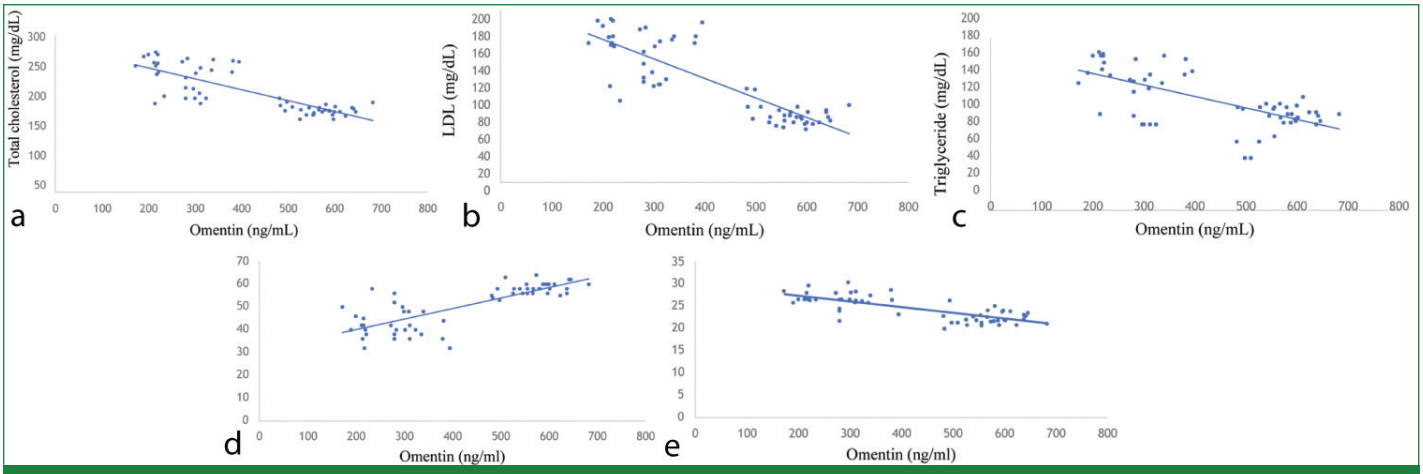
The mean value of omentin in males was 457.6 ng/mL, while in females, it was 393.4 ng/mL. The mean value of serum omentin-1 in the diabetic population was significantly lower than in the non diabetic controls. Additionally, the mean values of cholesterol, LDL, and triglycerides in the diabetic population were significantly higher than in the non diabetic population. Moreover, the mean HDL concentration in the diabetic population was significantly lower than in the non diabetic population. Lastly, the mean BMI in the diabetic population was significantly higher than in the non diabetic population [Table/Fig-2].

Analyte	Diabetic (n=30) (mean±2SD)	Non Diabetic (n=30) (mean±SD)	T-test	p-value
Omentin-1 (ng/mL)	273.37±60.18	573.4±51.6	20.73	<0.001
FBS (mg/dL)	197.5±54.618	91.83±6.092	10.531	<0.001
Total cholesterol (mg/dL)	225.83±31.19	157.47±9.205	11.515	<0.001
LDL (mg/dL)	152.9±28.053	78.17±11.136	13.562	<0.001
Triglyceride (mg/dL)	151.7±27.739	106.8±17.407	7.51	<0.001
HDL (mg/dL)	42.5±6.501	58.2±2.644	12.253	<0.001
HbA1c (%)	8.30±1.76	5.13±0.31	9.709	<0.001
BMI	26.66±1.77	22.36±1.43	10.345	<0.001

[Table/Fig-2]: Comparison of analytes between T2DM and non diabetics[1].
Comparison of analytes between T2DM and non diabetics using student t-test; FBS: Fasting blood sugar; LDL: Low density lipoprotein; HDL: High density lipoprotein; HbA1c: Glycated haemoglobin; BMI: Body mass index

Correlation	Cases and controls	
	Pearson correlation r value	p-value
Omentin-1 with Total cholesterol	-0.811	<0.001
Omentin-1 with LDL	- 0.845	<0.001
Omentin-1 with Triglyceride	-0.667	<0.001
Omentin-1 with HDL	0.796	<0.001
Omentin-1 with BMI	-0.771	<0.001

[Table/Fig-3]: Correlation of omentin 1 level with lipid profile and BMI in diabetic cases and non diabetic controls.



[Table/Fig-4]: a) Scatter plot showing correlation between omentin and total cholesterol; b) Scatter plot showing correlation of omentin with LDL; c) Scatter plot showing correlation of omentin with triglyceride; d) Scatter plot showing correlation of omentin with HDL; e) Scatter plot showing correlation of omentin with BMI.

Serum omentin showed a significantly strong negative correlation with serum total cholesterol, LDL, and triglycerides in both diabetic cases and non diabetic controls. Furthermore, serum omentin exhibited a significant positive correlation with serum HDL in both diabetic cases and non diabetic controls. Serum omentin also showed a significantly strong negative correlation with BMI in both diabetic cases and non diabetic controls. The results have been summarised in [Table/Fig-3,4a-e].

DISCUSSION

The present study demonstrated that the mean value of serum omentin-1 in the diabetic population was significantly lower than in non diabetic controls. This finding is consistent with previous studies conducted by El-Mesallamy HO et al., which also reported significantly reduced omentin levels in the diabetic population compared to non diabetic individuals [9]. A review conducted by Escote X et al. also supported these findings, showing a significant reduction in omentin levels in the diabetic population [11]. Similarly, Elsaid NH et al. observed significantly reduced serum omentin levels in diabetic patients compared to healthy controls [12], and Pan HY et al. reported a similar reduction in omentin levels in diabetic individuals compared to healthy controls [13].

Furthermore, the mean values of total cholesterol, LDL, and triglycerides in the diabetic population were significantly higher than in the non diabetic population, and these values exhibited a significant strong negative correlation with omentin. El-Mesallamy HO et al. observed significantly higher total cholesterol and LDL levels in the diabetic population compared to non diabetic individuals, and these values showed a strong negative correlation with serum omentin [9]. Herder C et al. reported a significant negative correlation between triglycerides and serum omentin levels [14]. Zengi S et al. also found a significant negative correlation between both triglycerides and total cholesterol with serum omentin levels [15]. Moreno-Navarrete JM et al. observed a significant negative correlation between triglycerides and serum omentin levels [16].

Moreover, the mean HDL concentration in the diabetic population was significantly lower than in the non diabetic population, and these values showed a significant positive correlation with omentin. De Souza Batista CM et al., Herder C et al., and Urbanová M et al. all observed a significant positive correlation between HDL and serum omentin levels [8,14,17].

The effects of diabetes mellitus (DM), including dyslipidemia and long-term organ damage, are attributed to defects in insulin secretion, insulin action, or both [18]. Specifically, defects in insulin action can be attributed to reduced secretion of insulin-sensitising adipokines, such as omentin, with obesity being a major contributing factor. Studies have shown that insulin and glucose significantly decrease the expression of omentin mRNA and protein production in adipose tissue [12]. Reduced levels of omentin-1 in diabetic patients can lead to a decrease in insulin-stimulated glucose uptake in insulin-sensitive tissues, contributing to insulin resistance and its associated consequences [19,20]. Additionally, omentin-1 has been found to stimulate 5-AMP-activated protein kinase, which acts as an inhibitor of cholesterol synthesis [12]. Therefore, reduced omentin levels in DM can potentially lead to increased cholesterol levels. Dyslipidemia, a major predictor of cardiovascular complications in type 2 diabetes mellitus (T2DM), is considered to be the most significant consequence of reduced omentin levels and insulin resistance. In conditions related to obesity, increased levels of free fatty acids can decrease the activity of lipoprotein lipase in adipose tissue and skeletal muscle, inhibit lipolysis of chylomicrons, and increase the synthesis of very low-density lipoproteins (VLDL) in the liver, leading to hypertriglyceridemia [21]. The relationship between omentin-1 and high-density lipoprotein cholesterol (HDL-C) may be attributed to impairments in insulin signaling resulting from changes in circulating omentin-1 levels [22]. Hypertriglyceridemia triggers the exchange of triglycerides for cholesterol esters between triglyceride-rich lipoproteins (VLDL, intermediate-density lipoprotein) and lipoproteins that are relatively richer in cholesterol esters (HDL), resulting in decreased HDL-cholesterol concentration [23].

According to the criteria set by the World Health Organisation (WHO), the diabetic cases in the study population are considered to be obese, while the non diabetic controls fall within normal BMI ranges [24]. This supports the conclusion that excess adiposity leads to an imbalanced production of adipocytokines, which are responsible for the development of obesity-related metabolic disorders by affecting lipid and glucose homeostasis, as well as inflammatory pathways that may contribute to the development of DM and dyslipidemia [25]. Consequently, omentin can be used as a biomarker for insulin resistance and to predict cardiovascular complications in T2DM.

Limitation(s)

This study focuses solely on the relationship between omentin and type 2 diabetes mellitus (T2DM). It does not explore the relationship between different adipokines, their interplay, or their association with the inflammatory state in T2DM. Understanding these relationships would provide deeper insights into the molecular basis and complications of the disease. Additionally, this study does not investigate how omentin levels may vary in response to treatment for insulin resistance.

CONCLUSION(S)

In this study, the authors observed significantly lower serum omentin-1 levels in the diabetic population compared to the non diabetic population. They also found a significant negative

correlation between serum omentin levels and total cholesterol, LDL, triglycerides, and BMI, as well as a significant positive correlation with HDL in both diabetic and non diabetic controls. However, further studies are needed to explore the relationships between different adipocytokines, investigate how omentin levels vary in response to insulin resistance treatment, and determine its utility as a biomarker for assessing insulin sensitivity and predicting cardiovascular complications.

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