Relationship between omentin-1 and carotid intima thickness in type 2 diabetes mellitus

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Introduction

Diabetes mellitus (DM) is a widespread health problem. The prevalence of DM is expected to rapidly increase from 171 million (2.8% of the world’s population) in 2000 to 366 million (4.4% of the world’s population) by 2030 [1]. Individuals with DM have two- to three-fold increased risk for myocardial infarction or stroke compared with individuals without DM [2].

However, the underlying mechanisms linking type 2 DM with cardiovascular disease remain unclear. Recently, evidence has shown that some adipokines are major regulators of insulin resistance and direct mediators of endothelial dysfunction and macrophage infiltration of vessel walls [3].

Omentin-1 (also called inteletin-1, endothelial lectin HL-1, and intestinal lactoferrin receptor) has been identified as a major visceral (omental) fat secretory adipokine [4].

Mature omentin is a secretory glycoprotein consisting of 295 amino acids and N-linked oligosaccharides, and its basic structural unit is a 120 kDa homotrimer in which 40 kDa polypeptides are bridged by disulfide bonds [4].

Omentin-1 is a new type of Ca2+-dependant lectin, with an affinity for galactofuranosyl residue-containing constituents of pathogens and dominant immunogens [5]. Therefore, it was suggested that a biological function of omentin-1 (inteletin-1) is the specific recognition of pathogens and bacterial components, an important role in the innate immune response to parasitic infection [6].

Further, several studies have shown that omentin gene expression is altered by inflammatory state and obesity [7]. The gene is located in the 1q 22-q23 chromosomal region, which has previously been linked to type 2 DM in several populations [8,9]. It has also been suggested to inversely affect atherosclerosis. Carotid intima media thickness (CIMT) is a useful marker for subclinical atherosclerosis and is significantly correlated with various metabolic risk factors.

Patients and methods

Sixty participants were enrolled in the study: 30 patients with type 2 DM and 30 controls with normal glucose levels. Patients were classified into group I, which included 15 patients with CIMT greater than 0.9 mm, and group II, including 15 patients with CIMT less than 0.9 mm. All groups were subjected to full medical history taking, clinical examination, and assessment of fasting blood glucose levels, lipid profile, and serum omentin-1 levels using enzyme-linked immunosorbent assay. BMI and CIMT were also assessed using color Doppler ultrasound.

Results

Serum omentin-1 levels were significantly decreased in patients with type 2 DM compared with controls and were further decreased in patients with increased CIMT. Omentin-1 levels were negatively correlated with fasting blood sugar, BMI, waist circumference, lipid profile, and CIMT, and were positively correlated with high-density lipoprotein, with r-values of −0.72, −0.9, and −0.81 for fetal bovine serum, BMI, and CIMT, respectively; −0.58, −0.70, and −0.49 for triglyceride, low-density lipoprotein, and cholesterol, respectively; and +0.66 for high-density lipoprotein.

Conclusion

Serum omentin-1 level is decreased in type 2 DM patients and is negatively correlated with CIMT and BMI. Hence, omentin-1 could serve as a protective marker and predictor for cardiovascular disease. Further study needed to show whether omentin-1 is considered as a risk factor for DM.

Keywords:
carotid intima media thickness, omentin-1, type 2 diabetes mellitus

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Introduction

Omentin-1 is a novel adipokine that has a pivotal role in modulating insulin sensitivity, immunity, and inflammation. Adipokines contribute directly to the atherosclerotic process. The current study was conducted to evaluate the serum omentin-1 level in type 2 diabetes mellitus (DM) patients and to evaluate its relationship with carotid intima media thickness (CIMT).

Patients and methods

Sixty participants were enrolled in the study: 30 patients with type 2 DM and 30 controls with normal glucose levels. Patients were classified into group I, which included 15 patients with CIMT greater than 0.9 mm, and group II, including 15 patients with CIMT less than 0.9 mm. All groups were subjected to full medical history taking, clinical examination, and assessment of fasting blood glucose levels, lipid profile, and serum omentin-1 levels using enzyme-linked immunosorbent assay. BMI and CIMT were also assessed using color Doppler ultrasound.

Results

Serum omentin-1 levels were significantly decreased in patients with type 2 DM compared with controls and were further decreased in patients with increased CIMT. Omentin-1 levels were negatively correlated with fasting blood sugar, BMI, waist circumference, lipid profile, and CIMT, and were positively correlated with high-density lipoprotein, with r-values of −0.72, −0.9, and −0.81 for fetal bovine serum, BMI, and CIMT, respectively; −0.58, −0.70, and −0.49 for triglyceride, low-density lipoprotein, and cholesterol, respectively; and +0.66 for high-density lipoprotein.

Conclusion

Serum omentin-1 level is decreased in type 2 DM patients and is negatively correlated with CIMT and BMI. Hence, omentin-1 could serve as a protective marker and predictor for cardiovascular disease. Further study needed to show whether omentin-1 is considered as a risk factor for DM.

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Therefore, in this study we measured serum omentin levels in patients with DM to determine whether there is relationship between omentin levels and CIMT, to determine whether serum omentin levels are correlated with other risk factor; for example, BMI, waist circumference, and lipid profile, and to determine whether omentin-1 can be considered as a protective and predictive marker for cardiovascular diseases in diabetic patients.

Patients and methods
The present study is a hospital-based case–control study carried out over a period of 6 months, from December 2012 to May 2013, on a total sample of 60 participants (30 patients with type 2 DM and 30 age-matched and sex-matched controls with normal glucose tolerance levels). Patients were selected randomly from those admitted to the Internal Medicine Department of Al Zahraa University Hospital.

Patients with a history of cardiovascular disease (myocardial infarction, unstable angina, stroke), stage 2 hypertension (>160/100 mmHg), malignancy, renal diseases, hepatic diseases, and inflammatory conditions such as vasculitis or aortitis were excluded.

Patients were classified into two groups:

Group I: included 15 patients with type 2 DM with CIMT greater than 0.9 mm.

Group II: included 15 patients with type 2 DM with CIMT less than 0.9 mm.

Thirty participants with normal glucose levels served as controls.

The patients were subjected to biochemical tests. Fasting blood glucose levels were determined: diabetes was defined by a fasting plasma glucose value of 7 mmol/l or higher (126 mg/dl). All participants in the control group were subjected to a 75 g oral glucose loading test to exclude those with prediabetes and those with undiagnosed diabetes. Serum cholesterol, high-density lipoprotein (HDL), low-density lipoprotein (LDL), and triglyceride levels were determined colorimetrically on Hitachi 736 (Hitachi, Tokyo Japan). Serum concentration of omentin-l was determined using enzyme-linked immunosorbent assay kits.

Principle of the assay
The kit assays the level of human omentin-1 in samples on the basis of the double-antibody sandwich enzyme-linked immunosorbent assay technique. The sample containing omentin-1 is added to a well of a microtitre plate preloaded with human omentin-1 monoclonal antibodies and is incubated. Then add omentin-1 antibodies labeled with biotin and combined with streptavidin HRP to form Immune Complex.

Measurement of carotid intima media thickness
CIMT is measured manually using a high-resolution B-mode tomographic ultrasound system (MyLab50, Esaote, Italy) with a 7.5–10 MHz linear transducer. Precision of the CIMT measurement is 0.01 mm. CIMT was measured on the fall wall of the right and left common carotid arteries, 1.5 cm proximal to the bifurcation. The transducer was manipulated so that the lumen diameter was maximized in the longitudinal plane. The mean CIMT value of the right and left common carotid arteries was used for analysis. A carotid plaque was defined as a focal structure protruding into the arterial lumen, with a thickness 1.3 mm or higher; the mean thickness is 0.08 mm.

Body mass index
BMI was calculated as weight/height$^2$ (kg/m$^2$), and waist circumference (WC) was measured at the midpoint between the lower border of the rib cage and the iliac crest.

Statistical design
Data collected were reviewed. Coding and statistical analysis of collected data were carried out using SPSS program (version 15; SPSS Inc., Chicago, Illinois, USA).

1) Descriptive statistics:
   (a) Central tendency and dispersion of quantitative data were expressed as mean and SD.
   (b) Qualitative data were expressed in terms of frequency of occurrence.

2) Analytical statistics:
   (a) Groups were compared using the following tests:
      (i) $\chi^2$-test: for comparison of qualitative data.
      (ii) Student’s $t$-test: for comparison of quantitative data between two groups.
      (iii) Pearson’s test: Pearson’s correlation coefficient was used to determine the association between two variables.
   (b) The level of significance was taken at a $P$-value of less than 0.05.
   (c) The results are represented in tables and graphs.

Results
This study was conducted on 30 patients with type 2 DM and 30 healthy participants. Patients included
13 men and 17 women; their ages ranged from 39 to 48 years, with a mean ± SD age of 43.20 ± 3.26 years. They were classified as follows:

Group I: included 15 diabetic patients (seven male and eight female) with CIMT greater than 0.9 mm; their ages ranged from 39 to 48 years, with a mean ± SD age of 43.73 ± 3.15 years.

Group II: included 15 diabetic patients (six male and nine female) with CIMT less than 0.9 mm; their ages ranged from 38 to 48 years, with a mean ± SD age of 42.67 ± 3.39 years.

Controls: included 12 men and 18 women; their ages ranged from 35 to 47 years, with a mean ± SD age of 41.40 ± 3.31 years.

On comparing the patient group with the control group, an insignificant difference in age and sex was found between them. However, a significant increase in the fasting bovine serum (FBS) level was found in the patient group (151.87 ± 19.83) compared with the control group (93.73 ± 7.78). In addition, anthropometric measures such as BMI and waist circumference were also significantly higher among patients (30.10 ± 4.44 and 98.20 ± 11.65, respectively) compared with controls (23.47 ± 2.35 and 82.60 ± 5.05, respectively). With regard to the lipid profile, a significant increase in cholesterol, triglyceride, and LDL levels was recorded in the patient group (179.48 ± 43.07, 173.52 ± 57.82, and 96.55 ± 22.69, respectively) compared with the control group (118.87 ± 33.39, 97.00 ± 12.02, and 32.87 ± 7.31, respectively), whereas the HDL level was significantly lower in the patient group (34.13 ± 6.642) compared with the control group (44.93 ± 6.59). Serum omentin-1 level was significantly decreased in the patient group (6.75 ± 3.30) compared with the control group (11.92 ± 1.34). CIMT was significantly higher in the patient group (0.93 ± 0.20) compared with the control group (0.64 ± 0.08) (Table 1).

On comparing patients of group I with those of group II, there was an insignificant difference in age and sex between the groups. However, a significant increase in FBS levels was found among group I patients (160.67 ± 21.48) compared with group II patients (143.07 ± 13.69). In addition, BMI and waist circumference were significantly increased in group I patients (31.93 ± 3.86 and 105.60 ± 8.63, respectively) compared with group II patients (27.60 ± 2.94 and 90.80 ± 9.45, respectively). Group I also showed higher cholesterol, triglyceride, and LDL levels (180.93 ± 49.95, 178.80 ± 65.40, and 101.73 ± 18.93, respectively) compared with group II patients (178.20 ± 38.05, 173.80 ± 48.55, and 95.73 ± 20.22, respectively); however, the differences were insignificant. In contrast, HDL levels were significantly lower among group I patients (30.67 ± 2.92) compared with group II patients (37.20 ± 7.78). With regard to serum omentin-1 levels, there was significant decrease in the level among group I patients (3.96 ± 1.88) compared with group II patients (9.55 ± 1.53); the reverse trend was seen for CIMT (1.06 ± 0.22 for group I patients and 1.80 ± 0.07 for group II patients), with a statistically significant difference (Table 2).

On comparing patients of group I with controls, there was an insignificant difference in age and sex between both groups. There was a significant increase in the FBS level in group I patients (160.67 ± 21.48) compared with the control group (93.73 ± 7.78). In addition, BMI and waist circumference were also significantly higher in the patient group (30.10 ± 4.44 and 98.20 ± 11.65, respectively) compared with the control group (23.47 ± 2.35 and 82.60 ± 5.05, respectively). With regard to the lipid profile, a significant increase in cholesterol, triglyceride, and LDL levels was recorded in the patient group (179.48 ± 43.07, 173.52 ± 57.82, and 96.55 ± 22.69, respectively) compared with the control group (118.87 ± 33.39, 97.00 ± 12.02, and 32.87 ± 7.31, respectively), whereas the HDL level was significantly lower in the patient group (34.13 ± 6.642) compared with the control group (44.93 ± 6.59). Serum omentin-1 level was significantly decreased in the patient group (6.75 ± 3.30) compared with the control group (11.92 ± 1.34). CIMT was significantly higher in the patient group (0.93 ± 0.20) compared with the control group (0.64 ± 0.08) (Table 1).

| Table 1 Comparative statistics between patients and controls |
|-------------------------------------------------------------|
| **Patients** | **Controls** | **Significance test** | **P-value** |
| Age (years) | Mean ± SD | 43.20 ± 3.26 | 41.40 ± 3.31 | t = 1.73 | 0.090 |
| | Range | 39–48 | 35–48 |
| Sex (N = 30) [n (%)] | | | | | |
| Male | 13 (43.3) | 12 (40.0) | χ² = 0.06 | 0.793 |
| Female | 17 (56.7) | 18 (60.0) |
| Fasting blood sugar (mean ± SD) | 151.87 ± 19.83 | 93.73 ± 7.78 | t = 10.87 | 0.000* |
| BMI (mean ± SD) | 30.10 ± 4.44 | 23.47 ± 2.35 | t = 5.39 | 0.000* |
| Waist circumference (mean ± SD) | 98.20 ± 11.65 | 82.60 ± 5.05 | t = 4.87 | 0.000* |
| Cholesterol (mean ± SD) | 179.48 ± 43.07 | 118.87 ± 33.39 | t = 4.46 | 0.000* |
| TG (mean ± SD) | 173.52 ± 57.82 | 97.00 ± 12.02 | t = 4.84 | 0.000* |
| HDL (mean ± SD) | 34.13 ± 6.642 | 44.93 ± 6.59 | t = -5.20 | 0.000* |
| LDL (mean ± SD) | 96.55 ± 22.69 | 32.87 ± 7.31 | t = 10.16 | 0.000* |
| Omentin-1 (mean ± SD) | 6.75 ± 3.30 | 11.92 ± 1.34 | t = -5.83 | 0.000* |
| CIMT (mean ± SD) | 0.93 ± 0.20 | 0.64 ± 0.08 | t = 4.87 | 0.000* |

CIMT, carotid intima media thickness; HDL, high-density lipoprotein; LDL, low-density lipoprotein; TG, triglyceride. *Significant difference (P < 0.05).
in group I patients (31.93 ± 3.86 and 105.60 ± 8.63, respectively) compared with controls (23.47 ± 2.35 and 82.60 ± 5.05, respectively). A significant increase in cholesterol, triglyceride, and LDL levels was recorded among group I patients (180.93 ± 49.95, 178.80 ± 65.40, and 101.73 ± 18.93, respectively) compared with controls (118.87 ± 33.39, 97.00 ± 12.02, and 32.87 ± 7.31, respectively), whereas HDL levels were significantly lower among group I patients (30.67 ± 2.92) compared with controls (44.93 ± 6.59). Serum omentin-1 levels were significantly decreased among group I patients (3.96 ± 1.88) compared with controls (11.92 ± 1.34). CIMT was significantly higher among group I patients (1.06 ± 0.22) compared with controls (0.64 ± 0.08; Table 3).

On comparing group II patients with controls, there was an insignificant difference in age and sex between both groups. There was a significant increase in FBS levels among group II patients (143.07 ± 13.69) compared with controls (93.73 ± 7.78). In addition, BMI and waist circumference were also significantly higher among group II patients (27.60 ± 2.94 and 90.80 ± 9.45, respectively) compared with controls (23.47 ± 2.35 and 82.60 ± 5.05, respectively). A significant increase in cholesterol, triglyceride, and LDL levels was recorded among group II patients (178.20 ± 38.05, 173.80 ± 48.55, and 95.73 ± 20.22, respectively) compared with controls (118.87 ± 33.39, 97.00 ± 12.02, and 32.87 ± 7.31, respectively), whereas HDL levels were significantly lower among group II patients (30.67 ± 2.92) compared with controls (44.93 ± 6.59). Serum omentin-1 levels were significantly lower among group II patients (9.55 ± 1.53) compared with controls (11.92 ± 1.34), whereas CIMT was significantly higher among group II patients compared with controls (0.80 ± 0.07 and 0.64 ± 0.08, respectively; Table 4).

### Table 2 Comparative statistics between group I and group II patients

|                      | Group I patients | Group II patients | Significance test | P-value |
|----------------------|-----------------|------------------|------------------|---------|
| Age (years)          |                 |                  |                  |         |
| Mean ± SD            | 43.73 ± 3.15    | 42.67 ± 3.39     | t = 0.89         | 0.380   |
| Range                | 39–48           | 39–48            |                  |         |
| Sex (N = 15) [n (%)] |                 |                  |                  |         |
| Male                 | 7 (46.7)        | 6 (40.0)         | χ² = 0.13        | 0.712   |
| Female               | 8 (53.3)        | 9 (60.0)         |                  |         |
| Fasting blood sugar  |                 |                  |                  |         |
| (mean ± SD)          | 160.67 ± 21.48  | 143.07 ± 13.69   | t = 2.67         | 0.012*  |
| BMI (mean ± SD)      | 31.93 ± 3.86    | 27.60 ± 2.94     | t = 3.45         | 0.002*  |
| Waist circumference  |                 |                  |                  |         |
| (mean ± SD)          | 105.60 ± 8.63   | 90.80 ± 9.45     | t = 4.47         | 0.000*  |
| Cholesterol (mean ± SD) | 180.93 ± 49.95 | 178.20 ± 38.05   | t = 0.16         | 0.867   |
| TG (mean ± SD)       | 178.80 ± 65.40  | 173.80 ± 48.55   | t = 0.23         | 0.814   |
| HDL (mean ± SD)      | 30.67 ± 2.92    | 37.20 ± 7.78     | t = 3.04         | 0.005*  |
| LDL (mean ± SD)      | 101.73 ± 18.93  | 95.73 ± 20.22    | t = 0.83         | 0.409   |
| Omentin-1 (mean ± SD)| 3.96 ± 1.88     | 9.55 ± 1.53      | t = 8.91         | 0.000*  |
| CIMT (mean ± SD)     | 1.06 ± 0.22     | 0.80 ± 0.07      | t = 4.15         | 0.000*  |

CIMT, carotid intima media thickness; HDL, high-density lipoprotein; LDL, low-density lipoprotein; TG, triglyceride; *Significant difference (P < 0.05).

### Table 3 Comparative statistics between group I patients and controls

|                      | Group I patients | Control group | Significance test | P-value |
|----------------------|-----------------|---------------|------------------|---------|
| Age (years)          |                 |               |                  |         |
| Mean ± SD            | 43.73 ± 3.15    | 41.40 ± 3.31  | t = 1.97         | 0.058   |
| Range                | 39–48           | 35–48         |                  |         |
| Sex (N = 15) [n (%)] |                 |               |                  |         |
| Male                 | 7 46.7          | 12 40.0       | χ² = 0.18        | 0.669   |
| Female               | 8 53.3          | 18 60.0       |                  |         |
| Fasting blood sugar  |                 |               |                  |         |
| (mean ± SD)          | 160.67 ± 21.48  | 93.73 ± 7.78  | t = 11.32        | 0.000*  |
| BMI (mean ± SD)      | 31.93 ± 3.86    | 23.47 ± 2.35  | t = 7.83         | 0.000*  |
| Waist circumference  |                 |               |                  |         |
| (mean ± SD)          | 105.60 ± 8.63   | 82.60 ± 5.05  | t = 8.90         | 0.000*  |
| Cholesterol (mean ± SD) | 180.93 ± 49.95 | 118.87 ± 33.39| t = 4.00         | 0.000*  |
| TG (mean ± SD)       | 178.80 ± 65.40  | 97.00 ± 12.02 | t = 4.76         | 0.000*  |
| HDL (mean ± SD)      | 30.67 ± 2.92    | 44.93 ± 6.59  | t = 7.66         | 0.000*  |
| LDL (mean ± SD)      | 101.73 ± 18.93  | 32.87 ± 7.31  | t = 13.14        | 0.000*  |
| Omentin-1 (mean ± SD)| 3.96 ± 1.88     | 11.92 ± 1.34  | t = 13.31        | 0.000*  |
| CIMT (mean ± SD)     | 1.06 ± 0.22     | 0.64 ± 0.08   | t = 6.70         | 0.000*  |

CIMT, carotid intima media thickness; HDL, high-density lipoprotein; LDL, low-density lipoprotein; TG, triglyceride; *Significant difference (P < 0.05).
Omentin-1 levels had a significantly negative correlation with age, fasting blood sugar, BMI, waist circumference, cholesterol, triglyceride, and LDL levels, and CIMT. In contrast, they had a significantly positive correlation with HDL level. These correlations were mild (with age, cholesterol), moderate (with triglyceride and LDL levels, duration of the disease, and BMI), and marked (with FBS, CIMT, and waist circumference; Table 5 and Figs 1–3).

Discussion

Adipose tissue produces several hormones and cytokines termed adipokines, which have widespread effects on carbohydrate and lipid metabolism. They appear to play an important role in the pathogenesis of insulin resistance, diabetes, and atherosclerosis [10].

Omentin-1 is a newly identified adipokine that is highly and selectively expressed in visceral adipose tissue [5]. It enhances insulin sensitivity and glucose metabolism [4].

This study showed a significant decrease in serum omentin-1 levels among diabetic patients in comparison with the normal group ($P < 0.00$; Table 1), which agreed with several studies that reported that omentin-1 levels in type 2 DM patients are decreased compared with those in healthy controls [11,12].

This demonstrates the role of omentin in regulating metabolic function by stimulating glucose uptake in response to insulin in cultured adipocytes, suggesting that omentin has a beneficial effect on insulin sensitivity [4]. In addition, it has been reported that plasma omentin-1 levels declined after prolonged insulin–glucose infusion in healthy individuals. Further,

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**Table 4 Comparative statistics between group II patients and controls**

| Age (years) | Group II Patients | Control Group | Significance test | P value |
|-------------|-------------------|---------------|------------------|---------|
| Mean ± SD   | 42.67 ± 3.39      | 41.40 ± 3.31  | $t = 1.03$       | 0.310   |
| Range       | 39–48             | 35–48         |                  |         |
| Sex (N=15)  | [n (%)]           |               |                  |         |
| Male        | 6 40.0            | 12 40.0       | $\chi^2 = 0.000$| 1.000   |
| Female      | 9 60.0            | 18 60.0       |                  |         |
| Fasting blood sugar (mean ± SD) | 143.07 ± 13.69 | 93.73 ± 7.78 | $t = 12.09$ | 0.000* |
| BMI (mean ± SD) | 27.60 ± 2.94 | 23.47 ± 2.35 | $t = 4.86$ | 0.000* |
| Waist circumference (mean ± SD) | 90.80 ± 9.45 | 82.60 ± 5.05 | $t = 2.96$ | 0.000* |
| Cholesterol (mean ± SD) | 178.20 ± 38.05 | 118.87 ± 33.39 | $t = 4.53$ | 0.000* |
| TG (mean ± SD) | 173.80 ± 48.55 | 97.00 ± 12.02 | $t = 5.94$ | 0.000* |
| HDL (mean ± SD) | 37.20 ± 7.78 | 44.93 ± 6.59 | $t = 2.93$ | 0.007* |
| LDL (mean ± SD) | 95.73 ± 20.22 | 32.87 ± 7.31 | $t = 11.31$ | 0.000* |
| Omentin-1 (mean ± SD) | 9.55 ± 1.53 | 11.92 ± 1.34 | $t = 4.49$ | 0.000* |
| CIMT (mean ± SD) | 0.80 ± 0.07 | 0.64 ± 0.08 | $t = 5.58$ | 0.000* |

CIMT, carotid intima media thickness; HDL, high-density lipoprotein; LDL, low-density lipoprotein; TG, triglyceride; *Significant difference ($P < 0.05$).
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Treating the human omental tissue plants with glucose and insulin caused downregulation of omentin-1 expression [11]. We further demonstrated a significant decrease in the omentin-1 level in patients with an increase in CIMT, compared with the other diabetic group. This agrees with the findings of Yamawaki et al. [13], who reported that the plasma concentration of omentin is associated with endothelium-dependent vasodilation. In addition, Shibata et al. [14] found that circulating omentin levels were negatively correlated with CIMT, which is a marker of early atherosclerosis. Low levels of omentin are also associated with an increased prevalence of coronary artery disease [15].

Greulich et al. [16] found that omentin-1 was highly expressed and secreted by epicardial adipose tissue, had reduced expression among type 2 DM patients compared with controls, and was positively correlated with diastolic function. These data suggest that omentin may not only serve as a biomarker for metabolic disorders but also as a cardioprotective adipokine, and that a decrease in its level could contribute to the induction of cardiovascular dysfunction in DM patients.

Fantuzzi and Mazzone [3] reported that some adipokines, such as resistin, peptidin, and adiponectin, directly mediate vascular health by influencing the function of endothelial cells, arterial smooth muscle cells, and macrophages in the vessel wall.

Two other reports on the negative correlation of serum omentin-1 with CIMT have suggested cardioprotective and antiatherosclerotic roles of omentin-1 [17,14]. Hye Jin Yoo et al. [18] suggested that omentin-1 is regulated by inflammation, which is the most important factor linking type 2 DM with the progression of cardiovascular complications.

An experimental study by Somimaruyama et al. [19] to demonstrate the role of omentin in stimulating endothelial cell function and revascularization processes shows that systemic delivery of an adenoviral vector expressing omentin enhances blood flow recovery and capillary density in ischemic limbs of mice, which is accompanied by increased phosphorylation of Akt (also called protein kinase B) and endothelial nitric oxide synthase. In addition, treatment with omentin protein stimulated the phosphorylation of Akt and endothelial nitric oxide synthase in human umbilical vein endothelial cells, as well as stimulated the phosphorylation of AMP-activated protein kinase.

We found a significant difference in BMI and waist circumference between the diabetic group and the normal group. In addition, there was significant increase in BMI and waist circumference in group I compared with group II.

The negative correlation of omentin-1 level with BMI and waist circumference is explained by Desouza Batista et al. [7], who reported that among various human tissues, visceral adipose tissue produces a large amount of omentin and its gene expression in the visceral fat depot is reduced in obese individuals. Pan et al. [12] reported that low levels of circulating omentin are associated with obesity-induced metabolic dysfunction such as insulin resistance and glucose intolerance; this suggests that reduced levels of omentin may be an indicator of visceral fat accumulation, thereby being correlated with clustering of metabolic disorders.

Further, we found a significantly negative correlation between omentin-1 levels and cholesterol, triglyceride, and LDL levels, which agrees with the findings of Reishibata et al. [20], who reported that omentin-1 levels were significantly lower in patients with one
or more risk factors of metabolic disorders such as hyperlipidemia.

**Conclusion**

This study showed a significantly decreased omentin-1 level in type 2 DM patients as compared with controls, with a larger decrease among patients with increased CIMT. There was a negative correlation between omentin-1 level and other risk factors for cardiovascular and metabolic disorders such as BMI and lipid profile. Hence, we can consider omentin-1 levels to be predictive of atherosclerosis and early vascular complications of DM.

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