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ROLE OF ADIPOKINE OMENTIN-1 AND OXIDATIVE STRESS IN TYPE 2 DIABETES MELLITUS PATIENTS WITH METABOLIC SYNDROME – A CASE-CONTROL STUDY

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ABSTRACT

Background: In Type 2 Diabetes Mellitus, peripheral insulin resistance is largely due to a pronounced downregulation of insulin receptors. The condition is also characterized by lower circulating levels of adipokines such as omentin, which may contribute to disruptions in glucose metabolism. However, the exact regulatory mechanisms governing the expression and activity of these adipokines remain unclear. Various factors—including metabolic syndrome, obesity, chronic inflammation, insulin resistance, and oxidative stress—have been proposed, but their specific contributions are still under debate.
Objectives: The aim of this work is to evaluate the adipokine omentin-1 and oxidative stress in type 2 diabetes mellitus patients with metabolic syndrome and controls. And also find its correlation.
Materials and Methods: This case-control study was conducted on 294 individuals in the age group of 25-75 years, out of which 147 subjects were diagnosed with type 2 diabetes mellitus and 147 apparently healthy subjects serving as the control group. Participants underwent comprehensive history taking, detailed clinical examinations, anthropometric parameters, blood pressure measurements, and both routine and specific laboratory investigations, including assessments of diabetic markers, lipid profile, blood pressure, MDA, SOD and serum omentin-1 levels.
Results: The serum omentin-1 levels were significantly higher in the healthy controls in comparison with the case group.
Conclusion: Decreased omentin levels in the case group might have contributed to the development of insulin resistance and diabetes mellitus.
Keyword: Omentin-1, Adipokines, T2DM, metabolic syndrome, oxidative stress.

INTRODUCTION

Type 2 diabetes mellitus or non-insulin dependent diabetes mellitus (NIDDM) is a metabolic syndrome of multiple etiology characterised by chronic hyperglycaemia resulting from disturbances in glucose and lipid metabolism. Diabetes causes an array of microvascular and macrovascular complications and stroke with an increasing incidences worldwide. The various diabetic complications, b-cell dysfunction and worsening of glycaemic control is linked to increased reactive oxidative stress (ROS) and reactive nitrosative stress (RNS) owing to increased production of free radicals such as the nitric oxide, superoxide radical, hydrogen peroxide and the hydroxide radical and free radical induced lipid peroxidation and a deficiency in the antioxidant defence mechanisms. The lipid peroxidation of tissues which is primarily caused due to increased ROS, is thought to play an important role in the development of atherosclerosis and other microvascular complica-tions.[1] During the development of the disease, hyperglycemia causes increase in production of ROS in different tissues by forming advanced glycation end products. Hyperglycaemia-induced mitochondrial superoxide

production is the sole underlying mechanism by which it induces cellular damage. The antioxidant defence network maintains the mitochondrial level of ROS within balanced concentrations. However, in hyperglycemia environment, enhanced glucose flux through glycolysis and Krebs cycle causes an overdrive of mitochondrial electron transport chain (ETC) resulting in mitochondrial dysfunction and increased ROS formation.[2] Excessive levels of ROS leads to cellular dysfunction, altered cell cycle, altered cell-signalling, increased inflammation and also is linked to development of insulin resistance, impaired metabolic pathways, diabetes and cardiovascular disorders (CVD) through dysfunction of insulin secretion and metabolism. Antioxidant defence mechanisms involve both enzymatic and non-enzymatic strategies. Common antioxidants include vitamin A, C and E, non-enzymatic antioxidant and cofactor GSH (L-g-glutamyl-L-cysteinylglycine) and the enzymes superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GPx). They work in synergy with each other and against different types of free radicals.[3] The impairment of the endogenous antioxidant defence system is produced in many ways during chronic hyperglycaemia. Lipids with unsaturated double bonds are highly susceptible to damage by free radicals, this process is commonly known as lipid peroxidation and it has deleterious effects in the biological system and it has been strongly linked to diabetes pathogenesis and complications. Malondialdehyde (MDA) is formed as an end product of lipid peroxidation. Elevated MDA levels show adverse physiological consequences which include altering cell membrane structural integrity, inactivating membrane bound enzymes and cell surface receptors. MDA is involved in foam cell formation which leads to atherosclerosis and other cardiovascular diseases. The determination of MDA is an important parameter to evaluate in vivo lipid peroxidation.[4]

The adipokines secreted by the adipose tissue include visfatin and omentin-1.[5] Omentin, also known as omentin-1 or intelectin-1 (ITLN1), was first identified as an adipokine in the stromal-vascular fraction of visceral adipose tissue (VAT).[6] He. However, it is also expressed in other tissues such as the placenta, heart, and lungs.[7] The early cross-sectional investigations have found that higher levels of systemic omentin were linked to high level of adiponectin, high insulin sensitivity, low level of T2DM, and lower level of cardiovascular risk factors.[8] Omentin has been shown to have atheroprotective effects in mouse models.[9] On the other hand, longitudinal studies have found that both population-based cohorts and those with preexisting cardiovascular disorders had a higher rate of cardiovascular events and T2DM risk when their systemic omentin concentrations were higher.[10]

The purpose of this study is to see the role of omentin-1 and oxidative stress in Type 2 diabetes mellitus with metabolic syndrome patients and its correlation.

MATERIALS AND METHODS

Study Population: A total of 294 subjects were enrolled, including 147 cases and 147 healthy controls.

The diagnosis of MetS was based on the global consensus of MetS according to the 2005 International Diabetes Federation (Alberti et al., 2006). The patients must have central obesity (BMI is >30kg/m²), and any two out of the rest four factors of MetS diagnosis:

1. Elevated Triglycerides ≥ 150 mg/dL (or specific treatment for this lipid abnormality).
2. Decreased HDL- cholesterol value (< 40 mg/dL in males and < 50 mg/dL in females) or specific treatment for this lipid abnormality.
3. Raised blood pressure; systolic blood pressure ≥ 130 mm Hg or diastolic blood pressure ≥ 85 mmHg or (having been diagnosed with hypertension and were treated).
4. Elevated fasting plasma glucose (FPG; ≥126 mg/dl) or have been diagnosed with type II diabetes.

Participants with cases were included in the study according to the World Health Organization criteria.[11]

Inclusion Criteria

- Age group 25-75 years
- Type 2 diabetes mellitus with Mets.
- Volunteers who fulfilled above mentioned criteria were included in the study

Exclusion Criteria

- Patients with type one diabetic mellitus
- Smokers
- Pregnant women
- Chronic alcoholism

Ethical approval: The study was approved by Institutional Ethics Committee, SAI Tirupati University, Udaipur, Raj. (STU/IEC/2024/306).

Study Procedure

Blood sample collection

Ten millilitres of venous blood were collected after ten-twelve hours of fasting from each subject and then were then divided into two aliquots. For the first aliquot (2 ml), EDTA containing tube was used for the assessment of fasting plasma glucose, while for the second aliquot (8 ml), biochemistry tubes with a gel separator were used. After thirty minutes of an incubation period, the samples were centrifuged (at 1500 × g for fifteen minutes). A portion of the obtained serum was used for the estimation of lipid profile. The second portion of the serum used for the subsequent assay of omentin-1 was stored at 20°C.

Laboratory test: The estimation of plasma glucose was performed by a glucose oxidase method. The total serum cholesterol was estimated by enzymatic colorimetric tests with cholesterol esterase and cholesterol oxidase, while the serum triglycerides

evaluation was done by the enzymatic colorimetric tests with glycerol phosphate oxidase. The HDL-cholesterol was evaluated after precipitation of the apolipoprotein B-containing lipoproteins with phosphotungstic acid. The low-density lipoprotein cholesterol was calculated by the Friedewald formula.[12] Glycated hemoglobin (HbA1c) levels were quantified using kits procured from Beacon Diagnostics Pvt. Ltd. (India).

MDA and SOD was measured by colorimetric method.

Human omentin-1 was measured using the commercially available ELISA assays kit (Ray Biotechnology Company, U.S.A). All ELISA procedures were carried out according to the manufacturer's instructions.

Anthropometric Measurement

Weight (Kg), height (cm), and waist circumference (cm) were measured for all the participants. The body mass index (BMI) was calculated by dividing the weight (in kg) over the height square (m2), waist to height ratio (WHtR) as well as body fat percentage (BF %) was calculated according to the following equations:

WHtR = waist (cm)/ height (cm)

BF% = (1.20 x BMI) + (0.23 x age) - (10.8 x sex) - 5.4 ….

(Sex: male=1, female=2).[13]

Blood Pressure Measurement

Mercury sphygmomanometer was used to measure the Systolic blood pressure (SBP) and diastolic blood pressure (DBP) (mmHg) in a sitting position. The mean arterial pressure (MAP) was calculated from these measurements according to the equation below:

MAP = DBP + (SBP-DBP)/3.[14]

Statistical Analysis: SPSS version 20.0 software were used. T-test were used. p-value of <0.05 indicating the statistically significant. The Student's t test was used to assess the significance of difference in the levels of omentin-1 and chemerin between the cases and the control groups. The correlation analysis between serum omentin-1 and other biochemical parameters in the cases was performed by the correlation coefficient test.

RESULTS

Table 1: Anthropometric parameters of control subjects and cases (Mean ± SD)

Parameters	Mean±S.D		
	(cases)	(control)	p-value
Age (Y)	53.12±11.43	46.87±13.17	<0.001
WC (cm)	144.45±26.81	86.45±7.34	<0.001
BMI (Kg/m2)	30.52±7.61	24.27±2.86	<0.001
WtHR	0.907±0.179	0.52±0.053	<0.001
BF %	34.00±7.668	19.59±7.24	<0.001

Abbreviations: BMI-Basal metabolic rate, WtHR- Waist-Hip Ratio, WC-Waist circumference, BF%-Body fat percentage.

Table 1. demonstrate significant anthropometric differences between cases and controls. Cases showed substantially higher waist circumference (144.45±26.81 cm vs. 86.45±7.34 cm, p<0.001), BMI (30.52±7.61 kg/m² vs. 24.27±2.86 kg/m², p<0.001), and waist-to-hip ratio (0.907±0.179 vs. 0.52±0.053, p<0.001). Body fat percentage was markedly elevated in cases (34.00±7.668% vs. 19.59±7.24%, p<0.001). The mean age difference (53.12±11.43 years vs. 46.87±13.17 years, p<0.001) reflects the age-related nature of metabolic syndrome. These findings strongly support the central role of abdominal obesity and altered body composition in metabolic syndrome pathophysiology. The dramatic differences in waist circumference and waist-to-hip ratio particularly emphasize visceral adiposity as a key distinguishing feature between cases and controls. It demonstrate profound differences in clinical parameters between cases and controls.

Table 2: Blood pressure measurements and Biochemical parameters of control subjects and cases (Mean ± SD)

Parameters	Mean±S.D		
	(cases)	(control)	p-value
FBS (mg/dl)	174.77±49.54	100.63±9.67	0.05
PPBS (mg/dl)	269.09±67.54	137.43±17.44	0.05
HbA1C (%)	9.82±3.01	5.3±0.77	0.05
SBP (mmHg)	166.47±19.31	109.59±10.21	0.05
DBP (mmHg)	101.18±11.28	74.03±5.93	0.05
MAP (mmHg)	124.84±18.71	86.014±5.544	0.05
TC(mg/dl)	225.94±69.62	166.25±44.83	0.05
TG(mg/dl)	203.23±113.35	149.83±73.82	0.05
HDL(mg/dl)	43.03±13.08	46.96±11.32	0.05
LDL(mg/dl)	141.35±68.57	92.45±38.35	0.05
VLDL(mg/dl)	40.89±22.56	28.92±15.67	0.05

Abbreviations: FBS – Fasting blood sugar, PPBS- Post-prandial blood sugar, HbA1C- Glycated haemoglobin, TG- Triglyceride, TC- Total cholesterol, HDL- High density lipoproteins, LDL-Low density lipoproteins, VLDL-Very low density lipoproteins, SBP- Systolic blood pressure, DBP - Diastolic blood pressure, MAP- Mean arterial pressure.

Table 2. demonstrate significant biochemical parameters and blood pressure measurements differences between cases and controls. Glycemic parameters showed severe elevation in cases: FBS (174.77±49.54 mg/dl vs. 100.63±9.67 mg/dl), PPBS (269.09±67.54 mg/dl vs. 137.43±17.44 mg/dl), and HbA1C (9.82±3.01% vs. 5.3±0.77%). Blood pressure parameters were markedly elevated in cases: SBP (166.47±19.31 mmHg vs. 109.59±10.21 mmHg), DBP (101.18±11.28 mmHg vs. 74.03±5.93 mmHg). Lipid profiles revealed dyslipidemia in cases with elevated TC (225.94±69.62 mg/dl), TG (203.23±113.35 mg/dl), and LDL (141.35±68.57 mg/dl), while HDL was reduced (43.03±13.08 mg/dl).

Table 3: Oxidative stress, antioxidants and adipokine Omentin-1 of control subjects and cases (Mean ± SD)

Parameters	Mean±S.D		
	(cases)	(control)	p-value
MDA	5.02±1.01	3.12±0.99	0.05
SOD	1.05±0.60	2.08±0.94	0.05
Omentin-1 (ng/ml)	100.30±21.27	133.38±55.93	0.05

Abbreviations: MDA- Malondialdehyde, SOD- Super oxide dimutase

Table 3. demonstrate significant oxidative stress, antioxidants and adipokines differences between cases and controls. Oxidative stress markers showed increased MDA (5.02±1.01 vs. 3.12±0.99) and decreased SOD (1.05±0.60 vs. 2.08±0.94) in cases. Adipokine levels revealed reduced omentin-1 (100.30±21.27 ng/ml vs. 133.38±55.93 ng/ml) indicating dysregulated adipose tissue function.

Table 4: Correlation between adipokine omentin-1 and oxidative stress level

Correlation		Omentin-1 (ng/ml)
Anthropometric Parameters		
WC (cm)	Pearson Correlation	-.325
	p-value	.001
BMI (Kg/m2)	Pearson Correlation	-.146
	p-value	.012
BF %	Pearson Correlation	-.236
	p-value	.001
WtHR	Pearson Correlation	-.310
	p-value	.001
Glycaemic Parameters		
FBS (mg/dl)	Pearson Correlation	-.293
	p-value	.004
PPBS (mg/dl)	Pearson Correlation	-.316
	p-value	.023
HbA1C (%)	Pearson Correlation	-.300
	p-value	.001
Blood Pressure Parameters		
SBP (mmHg)	Pearson Correlation	-.299
	p-value	.002
DBP(mmHg)	Pearson Correlation	-.268
	p-value	.014
MAP (mmHg)	Pearson Correlation	-.296
	p-value	.022
Lipid Profile		
TC(mg/dl)	Pearson Correlation	-.103
	p-value	.077
TG(mg/dl)	Pearson Correlation	-.026
	p-value	.654
HDL(mg/dl)	Pearson Correlation	.002
	p-value	.973
LDL(mg/dl)	Pearson Correlation	-.083
	p-value	.157
VLDL(mg/dl)	Pearson Correlation	-.041
	p-value	.479
Oxidative stress		
MDA	Pearson Correlation	-.304
	p-value	.001
SOD	Pearson Correlation	0.024
	p-value	0.678
Adipokine		
Omentin-1 (ng/ml)	Pearson Correlation	1
	p-value	---

Abbreviations: FBS – Fasting blood sugar, PPBS- Post-prandial blood sugar, HbA1C- Glycated haemoglobin, BMI-Basal metabolic rate, WtHR- Waist-Hip Ratio, TG- Triglyceride, TC- Total cholesterol, HDL- High density lipoproteins, LDL-Low density lipoproteins, VLDL- Very low density lipoproteins, SBP- Systolic blood pressure,

DBP - Diastolic blood pressure, MAP- Mean arterial pressure, MDA- Malondialdehyde, SOD- Super oxide dimutase

Table 4. present correlation analysis between adipokines and various metabolic parameters in T2DM cases. Omentin-1 demonstrated significant negative correlations with anthropometric parameters including waist circumference (r=-0.325, p=0.001), BMI (r=-0.146, p=0.012), and waist-to-hip ratio (r=-0.310, p=0.001). Strong negative correlations were observed with glycemic parameters: FBS (r=-0.293, p=0.004), PPBS (r=-0.316, p=0.023), and HbA1C (r=-0.300, p=0.001). Blood pressure parameters showed significant negative associations: SBP (r=-0.299, p=0.002), DBP (r=-0.268, p=0.014), and MAP (r=-0.296, p=0.022). Negative correlations were observed with lipid profile parameters except HDL, Total cholesterol (r=-0.103, p=0.77), Triglyceride (r=-0.026, p=0.654), LDL (r=-0.083. p=0.157), VLDL (r=-0.041, p=0.479), HDL (r=0.002, p=0.973). Oxidative stress MDA showed significant negative correlation (r=-0.304, p=0.001) while SOD showed positive correlation (r=0.024, p=0.678).

These correlations support omentin-1's protective role in metabolic syndrome pathophysiology, providing evidence for their potential as biomarkers for metabolic dysfunction severity and therapeutic targets.

DISCUSSION

Omentin-1 is an anti-inflammatory adipokine produced mainly in visceral adiposity has insulin sensitivity effects and has linked to obesity and obesity related disorders as insulin resistance and diabetes. The exact physiological role of omentin-1 in glucose homeostasis is still understood. Circulating omentin-1 levels were documented to be negatively correlated to all anthropometric parameters, glycaemic parameters, Blood pressure parameters, lipid profiles except HDL. It is negatively correlated with MDA but positively correlated with SOD.[15,16]

Various studies have reported decreased omentin-1 levels in type2 diabetes, impaired glucose tolerance and obesity.[17,18] In our study we found statistically significant decrease in the mean serum omentin-1 levels in diabetic obese patients compared to control group. Our result supported by Egyptian study had done by El-Mesallamy et al,[19] who documented significant lower omentin-1 levels in diabetic patients as compared to healthy subjects. Similarly, Abd-Elbaky et al,[20] reported significant decreased levels of omentin-1 in diabetic Egyptian patients in comparison to controls.

The exact mechanisms leading to decreased omentin-1 levels in obesity and type 2 diabetes are still unknown.

Regarding the relation of omentin-1 to glycemic control we found strong negative correlations between fasting glucose, post-parandial glucose, HbA1c and serum omentin-1 within cases. Similarly, Nanda B et al, [23] reported negative correlation between omentin-1 and glycemic parameters. In contrast, Urbanova et al,[21] failed to find any significant association between serum omentin-1 and glucose levels in both obese and type 2 diabetic patients. Catoi et al,[22] also failed to find significant association between serum omentin-1 and glucose in morbidly diabetic patients but after using multiple regression analysis they found glucose might be an independent factor for the changes that might occur in the circulating omentin-1 levels.

Visceral adiposity has found to be more pathogenic than sub cutaneous adiposity through accelerating insulin resistance, type 2 diabetes and cardiovascular disorders.[16] Body fat distribution, waist and hip circumference were known to reflect the visceral adiposity. It has suggested that differences in adipose tissue distribution may influence the secretion of adipokines.[24] In our study we recruited BMI (mean ± SD 30.52±7.61 kg/m2) and we documented statistically significant difference in the anthropometric measurements between diabetic patients and controls ($P < 0.001$). This was supported by previous studies.[25]

Regarding the relation of serum omentin-1 to anthropometric parameters, we found significant negative correlations between BMI, waist circumference, body fat %, waist-hip ratio and serum omentin-1. Various studies,[26] also documented inverse correlation between BMI and omentin-1. On the contrary Auguet et al,[27] and Catoi et al,[22] didn't find significant correlation between omentin-1 and BMI in morbidly diabetic patients.

Our patients were diabetic and Mets and had insulin resistance and these factors were found to be associated with increased internal cholesterol synthesis with decreased cholesterol absorption when compared to healthy subjects.[28] In our study we found statistically significant increased levels in fasting lipid profile ($P < 0.001$) in patients compared to controls. Similarly De Souza Batista et al. had shown that visceral obesity strongly associated with dyslipidemia.[15] On the contrary the study by Hossein-nezhad et al,[25] didn't show any significant difference in lipid profile between the studied groups.

Regarding the relation between omentin-1 and fasting lipids we found negative correlations between serum omentin-1 and lipid profile parameters except HDL-C. Similarly the study done by Moreno-Navarrete et al,[16] had reported negative correlations between omentin-1 and (TG, TC and LDL-C). On the contrary, Hossein-Nezhad et al,[25] didn't find any significant correlations between omentin-1 and lipid profile in patients with metabolic syndrome. Also Abd ELbaky et al.[20] reported significant inverse relation between omentin-1 and TC in their diabetic groups. This may signify the role of omentin-1 in lipid metabolism where it has found that omentin 1can stimulates 5-AMP-activated protein kinase which acts as cholesterol synthesis inhibitor.[29,30] In

addition other studies,[21] showed positive correlation between omentin-1 and HDL. It had supposed that the relation between HDL-C and omentin-1 might be due to impairment in insulin signalling that might occur as a result of changes in circulating omentin-1levels.[31]

Hypertension has linked to type 2 diabetes and obesity. It had hypothesized that insulin has anti-natriuretic effect to be one of the leading factors for hypertension in these groups of patients.[32] In our study we found significant increase in mean systolic blood pressure in our patients compared to controls ($P < 0.01$). Also De Souza–Batista et al,[15] had found significant increase in mean SBP and DBP in patients compared to healthy subjects. In addition we found significant negative correlation between SBP and omentin-1. This result also documented by Auguet et al,[41] and Moreno-Navarrete et al.[33] On the contrary De Souza–Batista et al,[15] didn't document this association. Omentin-1 has known to have anti-atherogenic effects through its role in endothelial dysfunction,[33] preventing arterial calcification,[34] vasodilator effect on isolated blood vessels through inducing the secretion of endothelial nitric oxide,[35] inhibiting TNF-α which induce vascular endothelial inflammation,[36] and as inhibitor to the inflammatory cascade.[37] Accordingly these data document the possible role of omentin-1 in preventing diabetes and metabolic disorders.

T2DM is characterised by prolonged and increased intracellular and extracellular ROS generation. In this study ROS levels were studied by determining the MDA concentrations in serum. The ROS levels in diabetics were slightly higher than in controls although there was no significant difference between them. Free radicals attack membrane phospholipids causing lipid peroxidation and high levels of these oxidised products have been correlated with development of vascular complications. The high levels of MDA in serum can be linked to failure of antioxidant system to curb the deleterious action of free radicals and hence it serves as a reliable stress marker to assess free radical induced tissue dam age.[38] The increase in lipid peroxidation reflected by the increase in serum MDA levels in diabetics in the present study are in accordance with previous studies that hyperglycaemia increases lipid peroxidation from overproduction of free radicals in diabetics.[39] In our study we also found that lipid peroxidation MDA was significantly higher in cases while antioxidant SOD was lower in cases as compared to controls.[40] Kesavulu MM et al., also shows the same patterns regarding antioxidant. There was significant negative correlation in between omentin-1 and MDA while no any significant correlation in between omentin-1 and SOD.

CONCLUSION

Our study showed significant decrease in serum omentin-1 levels in type 2 diabetes mellitus with Mets patients. MDA was higher in cases while SOD was higher in control subjects. regarding our results the abnormalities in circulating omentin-1 may be used as a biomarker for diabetes, obesity and metabolic disorders. We will need to work on a large scale to detect the exact role of omentin-1 on glucose homeostasis and oxidative stress.

Conflict of interest: The authors declare no conflict of interest.

REFERENCES

1. I V, M H. Introductory Chapter. In: Lushchak V, editor. Oxidative Stress — Molecular Mechanisms and Biological Effects. InTech. 2012.
2. Brand MD. The sites and topology of mitochondrial superoxide production. Exp Gerontol. 2010; 45(7–8): 466–472, doi: 10.1016/j. exger.2010.01.003, indexed in Pubmed: 20064600.
3. Feher J, Csomos G, Vereckei A. Control of Free Radical Reactions in Biological Systems. In: Free Radical Reactions in Medicine. Berlin, Heidelberg: Springer Berlin Heidelberg. 1987: 11–17.
4. Noberasco G, Odetti P, Boeri D, et al. Malondialdehyde (MDA) level in diabetic subjects. Relationship with blood glucose and glycosylated hemoglobin. Biomed Pharmacother. 1991; 45(4–5): 193–196, indexed in Pubmed: 1932603.
5. Yang RZ, Xu AH, Pray J, Hong H, Jadhao S, Hansen B, Shuldiner AR, McLenithan JC, Gong DW. Cloning of omentin, a new adipocytokine from omental fat tissue in humans. In: Diabetes 52, A1–A1, 2003.
6. Herder C, Carstensen M, Ouwens DM. Anti-inflammatory cytokines and risk of type 2 diabetes. Diabetes Obes Metab 15, 39–50, 2013.
7. Jung CH, Jung SH, Kim BY, Kim CH, Kang SK, Mok JO. Association of serum omentin levels with cardiac autonomic neuropathy in patients with type 2 diabetes mellitus: a hospital-based study. Cardiovasc Diabetol 14, 140, 2015.
8. Herder C, Ouwens DM, Carstensen M, Kowall B, Huth C, Meisinger C, Rathmann W, Roden M, Thorand B. Adiponectin may mediate the association between omentin, circulating lipids and insulin sensitivity: results from the KORA F4 study. Eur J Endocrinol 172, 423–432, 2015.
9. Hiramatsu-Ito M, Shibata R, Ohashi K. Uemura Y, Kanemura N, Kambara T, Enomoto T, Yuasa D, Matsuo K, Ito M, Hayakawa S, Ogawa H, Otaka N, Kihara S, Murohara T, Ouchi N. Omentin attenuates atherosclerotic lesion formation in apolipoprotein E-deficient mice. Cardiovasc Res 110, 107–117, 2016
10. Wittenbecher C, Menzel J, Carstensen-Kirberg M, Biemann R, di Giuseppe R, Fritsche A, Isermann B, Herder C, Aleksandrova K, Boeing H, Weikert C, Schulze MB. Omentin-1, adiponectin, and the risk of developing type 2 diabetes. Diabetes Care 39, e79–e80, 2016.
11. Alberti KG and Zimmet ZP. 1998. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: Diagnosis and classification of diabetes mellitus, provisional report of a WHO consultation. Diabet Med. J, 15: 539– 553.
12. Friedewald W, Levy R and Fredrickson D.1972. Estimation of the concentration of low-density lipoprotein cholesterol in plasma without use of the ultracentrifuge. Clin Chem J, 18: 449-502.
13. Deurenberg P, Weststrate AJ and Seidell CJ.1991. Body mass index as a measure of body fatness: age- and sex specific prediction formulas. Br J Nutr., 65:105-114.
14. Bouchra L, Denis C and Christian R. 2005. Clinical review: Interpretation of arterial pressure wave in shock states. Crit Care J, 9:601-606.
15. de Souza Batista CM, Yang RZ, Lee MJ, Glynn NM, Yu DZ, Pray J, et al. Omentin plasma levels and gene expression are decreased in obesity. Diabetes 2007;56:1655–61.
16. Moreno-Navarrete JM, Catalan V, Ortega F, Gómez-Ambrosi J, Ricart W, Frühbeck G, et al. Circulating omentin

concentration increases after weight loss. Nutr Metab (Lond) 2010;7:27. http://dx.doi.org/10.1186/1743-7075-7-27.
17. Pan HY, Guo L, Li Q. Changes of serum omentin-1 levels in normal subjects and in patients with impaired glucose regulation and with newly diagnosed and untreated type 2 diabetes. Diabetes Res Clin Pract 2010;88(1):29–33. http://dx.doi.org/10. 1016/j.diabres.2010.01.013.
18. Cai RC, Wei L, Di JZ, Yu HY, Bao YQ, Jia WP. Expression of omentin-1 in adipose tissues in obese and type 2 diabetic patients. Zhonghua Yi Xue Za Zhi 2009;89(6):381–4.
19. El-Mesallamy HO, El-Derany MO, Hamdy NM. Serum omentin-1 and chemerin le vels are interrelated in patients with type 2 diabetes mellitus with or without ischaemic heart disease. Diabet Med 2011;28:1194–200. http://dx.doi.org/10. 1111/j.1464-5491.2011.03353.x.
20. Abd-Elbaky A, Abo-ElMatty DM, Mesbah NM, Ibrahim SM. Omentin and apelin concentrations in relation to obesity diabetes mellitus type two and cardiovascular diseases in egyptian population. Endocrinol Metab Int J 2015;2(2):18.
21. Urbanová M, Dostálová I, Trachta P, Drápalová J, Kaválková P, Haluzíková D, et al. Serum concentration and subcutaneous adipose tissue mRNA expression of omentin in morbid obesity and type II diabetes mellitus: the effect of very-low–calorie diet, physical activity and laparoscopic sleeve gastrectomy. Physiol Res 2014;63:207–18.
22. Cătoi AF, Suciu Ş, Pârvu AE, Copăescu C, Galea RF, Buzoianu AD, et al. Increased chemerin and decreased omentin-1 levels in morbidly obese patients are correlated with insulin resistance, oxidative stress and chronic inflammation. Clujul Med 2014;87:19–26.
23. Nanda B, Mahapatra S, Devi N, Swain S, Padhy RK, Rattanet R. Study of serum omentin-1 in relation to insulin resistance in type II diabetes mellitus. IOSR J Dental Med Sci 2015;14(12):12–21.
24. Wajchenberg BL. Subcutaneous and visceral adipose tissue: their relation to the metabolic syndrome. Endocr Rev 2000;21(6):697–738.
25. Hossein-nezhad A, Mirzaei K, Alatab S, Ahmadivand Z, Najmafshar A. Circulating omentin-1 in obesity and metabolic syndrome status compared to control subjects. Endocrinol Metabol Syndrome 2012;S1:008.
26. Tan Bee K, Adya Raghu, Farhatullah S, Lewandowski Kris C, O'Hare Paul, Lehnert Hendrik, et al. Omentin-1, a novel adipokine, is decreased in overweight insulin resistant women with polycystic ovary syndrome: ex vivo and in vivo regulation of omentin-1 by insulin and glucose. Diabetes 2008;57:801–8.
27. Auguet T, Quintero Y, Riesco D, Morancho B, Terra X, Crescenti A, et al. New adipokines vaspin and omentin. Circulating levels and gene expression in adipose tissue from morbidly obese women. BMC. Med Genet 2011;12:60. http://dx.doi. org/10.1186/1471-2350-12-60.
28. Simonen P, Gylling H, Howard A, Miettinen TA. Introducing a new component of the metabolic syndrome: low cholesterol absorption. Am J Clin Nutr 2000;72:82–8.
29. Kataoka Y, Shibata R, Ohashi K, Kambara T, Enomoto T, Uemura Y, et al. Omentin prevents myocardial ischaemic injury through AMP- activated protein kinase– and Akt-dependent mechanisms. J Am Coll Cardiol 2014;63:2722–33. http://dx.doi. org/10.1016/j.jacc.2014.03.032.
30. Yu D. Omentin activates AMP– activated protein kinase and plays a role in energy metabolism and immune response [PhD dissertation]. Molecular Medicine. University of Maryland: Baltimore, MD, USA; 2011.
31. Yan P, Liu D, Long M, Ren Y, Pang J, Li R. Changes of serum omentin levels and relationship between omentin and adiponectin concentrations in type 2 diabetes mellitus. Exp Clin Endocrinol Diabetes 2011;119:257–63.
32. Brands MW, Manhiani MM. Sodium-retaining effect of insulin in diabetes. Am J Physiol 2012;303(11):R1101–9.
33. Moreno-Navarrete JM, Ortega F, Castro A, Sabater M, Ricart W, Ferna'ndez-Real JM. Circulating omentin as a novel biomarker of endothelial dysfunction. Obesity 2011;19:1552–9. http://dx.doi.org/10.1038/oby.2010.351.
34. Yamawaki H, Tsubaki N, Mukohda M, Okada M, Hara Y. Omentin, a novel adipokine, induces vasodilation in rat isolated blood vessels. Biochem Biophys Res Commun 2010;393:668–72. http://dx.doi.org/10.1016/j.bbrc.2010.02.053.
35. Duan XY, Xie PL, Ma YL, Tang SY. Omentin inhibits osteoblastic differentiation of calcifying vascular smooth muscle cells through the PI3K/Akt pathway. Amino Acids 2011;41:1223. http://dx.doi.org/10.1007/s00726-010-0800-3.
36. Yamawaki H, Kuramoto J, Kameshima S, Usui T, Okada M, Hara Y. Omentin, a novel adipocytokine inhibits TNF-induced vascular inflammation in human endothelial cells. Biochem Biophys Res Commun 2011;408:339–43. http://dx.doi. org/10.1016/j.bbrc.2011.04.039.
37. Watanabe Kaho, Watanabe Rena, Konii Hanae, Shirai Remina, Sato Kengo, Matsuyama Taka-aki, et al. Counteractive effects of omentin-1 against atherogenesis. Cardiovasc Res 2016;110:118–28
38. Warnick GR, Knopp RH, Fitzpatrick V, et al. Estimating low-density lipoprotein cholesterol by the Friedewald equation is adequate for classifying patients on the basis of nationally recommended cutpoints. Clin Chem. 1990; 36(1): 15–19, indexed in Pubmed: 2297909.
39. Pasaoglu H, Sancak B, Bukan N. Lipid peroxidation and resistance to oxidation in patients with type 2 diabetes mellitus. Tohoku J Exp Med. 2004; 203(3): 211–218, indexed in Pubmed: 15240931.
40. Kesavulu MM, Rao BK, Giri R, et al. Lipid peroxidation and antioxidant enzyme status in Type 2 diabetics with coronary heart disease. Diabetes Res Clin Pract. 2001; 53(1): 33–39, indexed in Pubmed: 11378211.