

Serum Levels of Omentin in End-Stage Renal Disease Patients

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Key Words

Omentin · Hemodialysis · Diabetes mellitus · End-stage renal disease

Abstract

Introduction: Cardiovascular disease is the leading cause of morbidity and mortality in hemodialysis patients. Therefore, evaluation and prevention of cardiovascular diseases in end-stage renal disease (ESRD) patients are very important. The plasma level of omentin was found to be associated with different conditions such as insulin resistance. It is one of the novel adipokines synthesized mainly in the visceral adipose tissue. In this study, we aimed to investigate the level of omentin in patients with ESRD receiving hemodialysis. **Methods:** The study population consisted of 59 adult chronic hemodialysis patients (30 women and 29 men) and age-matched control subjects were selected from apparently healthy subjects (28 participants; 14 women and 14 men). Blood samples were obtained before the dialysis session. Omentin concentrations were determined by using enzyme-linked immunosorbent assay. **Results:** Plasma levels of omentin were found to be markedly higher in ESRD patients (606.6 ± 313.0 ng/ml) than in the control group (357.5 ± 147.4 ng/ml; $p < 0.0001$). Also, serum omentin levels were

found to be correlated with creatinine ($r = 0.333$, $p = 0.002$). **Conclusions:** Omentin levels were found to be elevated in patients with ESRD receiving hemodialysis. To the best of our knowledge, this is the first clinical study that demonstrated the association between omentin and ESRD.

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Introduction

The prevalence of end-stage renal disease (ESRD) has been increasing worldwide. It is an important health problem which causes high mortality and morbidity [1]. The main pathophysiologic mechanism underlying the high mortality of ESRD is premature atherosclerosis and chronic inflammation [2]. A large number of biomolecules have been shown to predict the atherosclerotic and inflammatory process in ESRD [3–6]. Adipose tissue produces a variety of proteins called adipokines. Thus it is accepted as an endocrine organ because it affects several organs and systems in the metabolism [7]. Besides inflammatory molecules such as leptin, resistin and visfatin in the family of adipokines, some useful molecules such as adiponectin are also secreted. In recent years, one of these useful adipokines called omentin has frequently

Table 1. Baseline characteristics of hemodialysis patients and control subjects

Parameters	Hemodialysis patients (n = 59)	Control subjects (n = 28)	p value
Age, years	56.8 ± 10.9	53.3 ± 8.8	0.143
Gender, male/female	29/30	14/14	0.941
Smoking, %	42.4	25.0	0.119
Systolic blood pressure, mm Hg	130.8 ± 20.4	119.6 ± 14.7	0.011
Diastolic blood pressure, mm Hg	78.6 ± 10.2	77.8 ± 9.1	0.730
Waist circumference, cm	96.1 ± 9.8	91.5 ± 14.4	0.080
BMI	26.4 ± 4.0	28.0 ± 5.6	0.133
Hemoglobin, g/dl	11.2 ± 1.2	13.9 ± 1.4	<0.001
Platelets, 10 ⁴ /μl	159.6 ± 55.1	244.6 ± 69.7	<0.001
Serum creatinine, mg/dl	6.3 ± 1.9	0.7 ± 0.08	<0.001
Fasting glucose, mg/dl	137.1 ± 49.5	98.5 ± 9.1	<0.001
HOMA-IR	3.7 ± 4.7	1.9 ± 3.0	0.076
TC, mg/dl	169.1 ± 38.8	204.3 ± 33.2	<0.001
LDL-C, mg/dl	89.9 ± 27.4	128.4 ± 33.2	<0.001
HDL-C, mg/dl	39.4 ± 9.4	44.3 ± 10.1	0.029
TG, mg/dl	200.3 ± 132.6	157.6 ± 62.2	0.109
C-reactive protein, mg/l	4.7 ± 4.1	2.8 ± 2.3	0.030
Plasma omentin, ng/ml	606.6 ± 313.0	357.5 ± 147.4	<0.001

HOMA-IR = Homeostatic model assessment of insulin resistance; HDL-C = high-density lipoprotein cholesterol; TG = triglycerides.

been studied. The plasma level of omentin (also named intelectin-1, intestinal lactoferrin receptor, endothelial lectin HL-1, or galactofuranose-binding lectin) was found to be associated with different conditions such as insulin resistance, diabetes mellitus, obesity, endothelial dysfunction and atherosclerosis. It is one of the novel adipokines synthesized mainly in the visceral adipose tissue [8, 9]. In metabolic syndrome omentin levels were found to be low. This condition may be related to the accelerated atherosclerosis in metabolic syndrome [10]. The appropriate treatment of diabetes and obesity had positive effects on the level of omentin in a limited number of studies [11, 12].

In this study, we aimed to investigate the level of omentin in patients with ESRD receiving hemodialysis.

Materials and Methods

Subjects

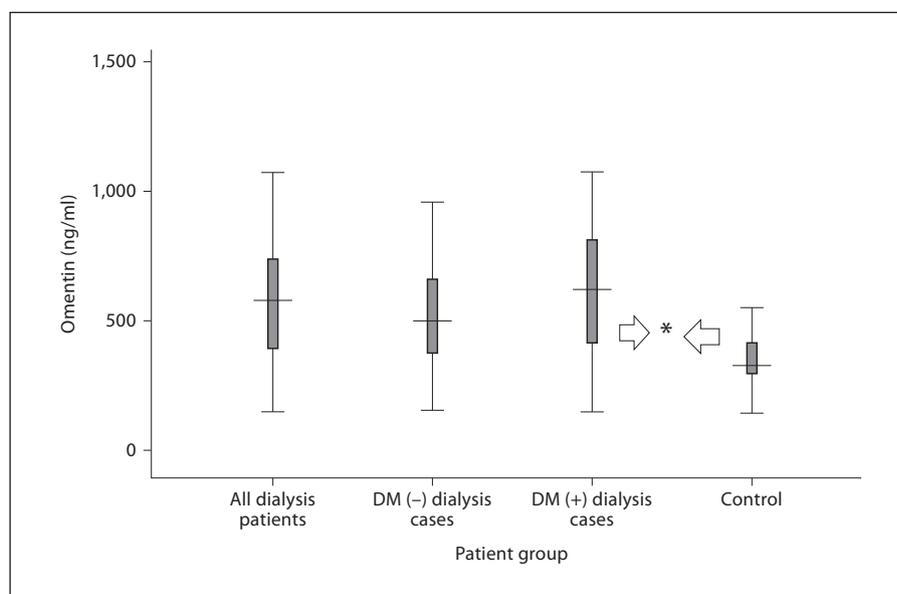
The study population consisted of 59 adult chronic hemodialysis patients (30 women and 29 men) who received regular (3 times a week) hemodialysis treatment at the hemodialysis unit of the Oray Dialysis Center. Age-matched control subjects were selected from apparently healthy subjects who visited the Izzet Baysal University

Hospital for a routine checkup (28 participants; 14 women and 14 men). Hemodialysis patients were dialyzed 3 times a week for 4 h with synthetic membranes (Xenium Dialyzer; Baxter Healthcare Corp., USA). The demographic and clinical characteristics of patients included in the study are shown in table 1. They continued with their regular medications, such as antihypertensives, recombinant human erythropoietin and phosphate binders. Etiologies of renal failure in the patients were hypertension (n = 30), diabetes mellitus (n = 10), chronic glomerulonephritis (n = 4), chronic interstitial nephritis (n = 3), Anderson-Fabry disease (n = 1), and undefined (n = 11). The median duration of hemodialysis at the time of the study was 42.8 months (range 4–183 months). The study protocol was approved by the ethics committee of our institution.

Methods

Anthropometric measurements obtained in this study included height, weight, body mass index (BMI), and waist circumference. BMI was calculated as predialysis body weight divided by height squared (kg/m²). Kt/V was calculated to assess the adequacy of dialysis. We measured abdominal circumference at the umbilical level. Blood pressure was measured with a sphygmomanometer in the sitting position on the right arm. Blood samples were obtained after an overnight fast before dialysis sessions, and the interval from the last HD session was 72 h for dialysis patients. Pregnancy and malignancy were excluded. Patients were free of acute infectious diseases and had received no antimicrobial agents during the preceding 2 weeks. Fasting blood samples were obtained for analysis of creatinine, total cholesterol (TC), triglycerides, high-density lipoprotein cholesterol, low-density lipopro-

Fig. 1. Serum omentin levels of the hemodialysis patients and the control group. They were found to be significantly higher in the hemodialysis patients than in the control group (* $p < 0.0001$).



tein cholesterol (LDL), uric acid, sodium bicarbonate, hemoglobin, albumin, calcium, inorganic phosphate, C-reactive protein (CRP), intact parathyroid hormone (iPTH), insulin and glucose with standard methods. Homeostatic model assessment was calculated with the formula [13]: $\text{insulin (mIU/l)} \times \text{glucose (mmol/l)} / 22.5$.

Omentin Measurement

Venous blood samples were centrifuged within 15 min of collection, at 2,750 g for 10 min, and the supernatant plasma was then transferred into polypropylene tubes at -80°C until the assays were determined. Omentin concentrations were determined using enzyme-linked immunosorbent assay according to the manufacturer's protocol. Plasma omentin levels were assessed using a commercial enzyme-linked immunosorbent assay kit (Bio Vendor, Brno, Czech Republic). The linear range of the assay was 0.50–64.0 mg/l. The inter- and intra-assay coefficients of variation were 4.4 and 3.2%, respectively.

Statistical Analysis

The normal distribution of all variables was tested using the Kolmogorov-Smirnov test. Continuous variables were described by the mean \pm standard deviation while interrelationships were examined using Pearson's correlation and t test. The Mann-Whitney U test was used to determine differences between nonparametric data. The associations between omentin levels and the indicated parameters were examined by single or multiple logistic regression analyses. Student's two-tailed t test was applied to compare continuous variables, and the χ^2 test was used for categorical data. All statistical analyses were performed using the SPSS 15.0 software (SPSS Inc., Chicago, Ill., USA). Results were considered significant when $p < 0.05$.

Results

Patient Characteristics

The clinical and demographic data of the patients are shown in table 1. There were no differences in age, sex, smoking, diastolic blood pressure and BMI between hemodialysis patients and controls. ESRD patients had significantly higher levels of omentin, systolic blood pressure, fasting glucose, creatinine, and CRP, and lower levels of TC, LDL, high-density lipoprotein cholesterol and hemoglobin than control subjects. Plasma levels of omentin were found to be markedly higher in ESRD patients (606.6 ± 313.0 ng/ml) than in the control group (357.5 ± 147.4 ng/ml; $p < 0.0001$) (table 1; fig. 1). Serum omentin had positive correlations with creatinine ($r = 0.333$, $p = 0.002$).

Serum Omentin Levels in Hemodialysis Group

Serum omentin levels and ESRD parameters are summarized in table 2. We did not find any association between serum omentin levels and other variables such as blood pressure, age, smoking, lipid parameters, adequacy of dialysis (Kt/V) and iPTH. However, there was a positive trend between plasma omentin levels and Kt/V ($r = 0.243$, $p = 0.06$) (fig. 2).

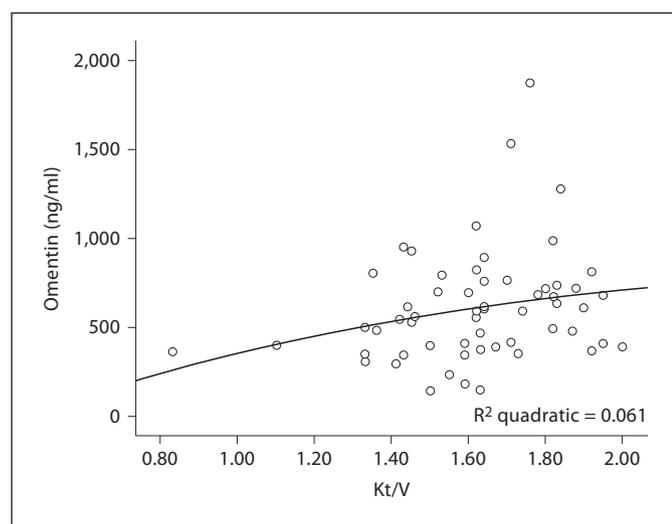
Omentin Levels in Diabetic and Nondiabetic Hemodialysis Subgroups

As shown in table 2, there was a significant difference in omentin serum levels between the nondiabetic ESRD

Table 2. Characteristics of ESRD patients

Parameters	Hemodialysis patients		p value
	diabetes (+) cases (n = 28)	diabetes (-) cases (n = 31)	
Age, years	61.1 ± 9.4	53.0 ± 10.8	0.003
Gender, male/female	13/15	16/15	0.693
HD duration, years	32.8 ± 25.2	51.9 ± 40.5	0.036
Systolic blood pressure, mm Hg	129.2 ± 19.8	132.2 ± 21.2	0.582
Diastolic blood pressure, mm Hg	76.7 ± 11.8	80.3 ± 8.3	0.188
Waist circumference, cm	99.9 ± 7.5	92.6 ± 10.4	0.004
BMI	27.4 ± 3.1	25.4 ± 4.5	0.057
Serum creatinine, mg/dl	5.7 ± 1.8	6.9 ± 1.9	0.012
Kt/V (per one dialysis session)	1.6 ± 0.2	1.6 ± 0.1	0.963
Serum HCO ₃ , mmol/l	19.9 ± 1.6	19.7 ± 0.9	0.501
Hemoglobin, g/dl	11.1 ± 1.1	11.2 ± 1.3	0.692
Intact serum PTH, pg/ml	248.8 ± 178.4	355.0 ± 183.2	0.028
Serum calcium, mmol/l	8.7 ± 0.5	8.8 ± 0.5	0.526
Serum phosphorus, mmol/l	4.2 ± 1.1	4.6 ± 1.1	0.281
Alkaline phosphatase, U/l	116.7 ± 53.0	115.1 ± 58.2	0.914
Serum uric acid, mg/dl	5.0 ± 0.9	5.4 ± 1.0	0.100
Albumin, g/dl	3.9 ± 0.4	4.0 ± 0.2	0.111
Glucose, mg/dl	169.7 ± 54.2	107.5 ± 14.5	<0.001
HbA1c, %	7.2 ± 1.2	-	-
HOMA-IR	4.3 ± 5.7	3.2 ± 3.6	0.373
TC, mg/dl	172.7 ± 43.2	165.8 ± 34.8	0.500
LDL, mg/dl	92.3 ± 25.8	87.8 ± 28.9	0.532
HDL, mg/dl	39.2 ± 8.8	39.5 ± 10.0	0.888
TG, mg/dl	219.3 ± 140.4	183.1 ± 125.0	0.300
C-reactive protein, mg/l	5.3 ± 4.2	4.1 ± 4.0	0.290
Plasma omentin, ng/ml	528.9 ± 235.1	676.7 ± 359.1	0.044

HD = Hemodialysis; HCO₃ = bicarbonate; PTH = parathyroid hormone; HbA1c = hemoglobin A1c; HOMA-IR = homeostasis model assessment of insulin resistance; HDL = high-density lipoprotein; TG = triglycerides.

**Fig. 2.** Correlation between Kt/V and serum omentin levels in hemodialysis patients.

group (n = 31) and the diabetic ESRD group (n = 28; p < 0.05). The diabetic ESRD subgroup had higher age (61.11 ± 9.47 vs. 53.00 ± 10.81, p = 0.003), shorter hemodialysis duration (p = 0.032), higher fasting blood glucose (p < 0.0001), lower creatinine (p < 0.011), lower iPTH (p = 0.028), and coronary artery disease (p = 0.005) and lower serum omentin (528.94 ± 235.10 vs. 676.76 ± 359.17, p = 0.044). We did not find any association between serum omentin levels and other ESRD variables in diabetic and nondiabetic subgroups. There were no significant correlations in hemoglobin, BMI, uric acid, diastolic and systolic blood pressure, TC, LDL cholesterol, triglycerides, Kt/V, and iPTH in the diabetic ESRD subgroup. There was a positive correlation between omentin and age (r = 0.382, p = 0.034) in the nondiabetic ESRD subgroup. Nondiabetic ESRD patients had significantly higher levels of omentin than control subjects (676.76 ± 359.17 vs. 357.54 ± 147.48, p < 0.0001, n = 28).

Relationship between Omentin and Clinical Parameters in Healthy Subjects

The correlation between the omentin levels and clinical parameters was evaluated. There was a negative correlation between omentin and TC, LDL and platelet counts ($r = -0.475$, $p < 0.011$; $r = -0.465$, $p < 0.013$; $r = -0.430$, $p < 0.022$) in healthy subjects.

Discussion

In the present study, we investigated the level of omentin in hemodialysis patients with ESRD. The main finding is that omentin levels in hemodialysis patients were found to be statistically higher than the levels of the control group. Besides, in the subgroup analysis, omentin levels of diabetic ESRD patients were found to be lower than of nondiabetic ESRD patients. To the best of our knowledge, this is the first clinical study that demonstrated the association between omentin and ESRD.

Systemic inflammation, accelerated atherosclerosis and insulin resistance are common pathogenic features of ESRD [14–17]. The relationship between inflammation, insulin resistance, atherosclerosis and omentin levels was demonstrated in many studies. Low omentin levels are associated with endothelial dysfunction, atherosclerosis and cardiovascular diseases. The plasma omentin level decreases in obesity, diabetes and impaired glucose tolerance [9, 16]. Omentin is a secreted factor that enhances the effect of insulin on glucose metabolism [18]. Nevertheless, the exact levels and biological effects of omentin in chronic kidney disease (CKD) are not sufficiently known. Also, the effects of CKD on omentin levels are not yet known. When we neglect the possible effects of CKD on omentin, the expected result of this study was the reduced levels of omentin in hemodialysis patients due to inflammation, insulin resistance and accelerated atherosclerosis. But unexpectedly, significantly higher levels of omentin were found in the hemodialysis group than in the control group. Then we evaluated the parameters that may affect omentin levels, such as the CRP level, insulin resistance, diabetes and documented coronary artery disease, in the entire study population. We found that these variables which could negatively affect the level of omentin were significantly higher in the hemodialysis group. In other words, although the parameters associated with a significant reduction of omentin were more prevalent, in our study, omentin was found to be higher in hemodialysis patients with ESRD. We considered that the reason of increased levels of omentin might be related to impaired renal clear-

ance of this molecule. So, omentin levels may be found to be higher in ESRD patients due to defective degradation and excretion. Besides, omentin is a relatively large protein, 40 kDa [19], which during hemodialysis may not be significantly cleared from plasma. Currently there is no study supporting our estimation related to omentin excretion, but generally most adipokines such as adiponectin, visfatin and resistin are elevated in patients with CKD, likely owing to decreased renal excretion [20–24].

But similarly, another important adipokine, adiponectin, has been shown to be eliminated or biodegraded through the renal route. Also, increased levels of adipokine were shown in parallel with deterioration of renal function [25–27]. Unfortunately, similar studies about omentin are not available yet.

Visceral obesity is associated with a higher risk of obesity-related comorbidities, such as insulin resistance and diabetes mellitus, than is peripheral obesity [28]. Omentin is a protein expressed and secreted from visceral but not subcutaneous adipose tissue that increases insulin sensitivity in human adipocytes [29]. In our study, when we compared omentin levels of diabetic and nondiabetic hemodialysis patients, omentin was found to be significantly lower in diabetic patients than in nondiabetics. Tan et al. [30] have found lower levels of omentin in type 1 diabetic patients than in control subjects. Similarly, in another study omentin levels were found to be lower in patients with newly diagnosed type 2 diabetes and impaired glucose regulation. In our study consistent results were found with these studies. The BMI of patients may affect the omentin levels. As in the other two studies, in our study, there was no significant difference between BMI of diabetic and nondiabetic dialysis patients [31]. Despite the significantly higher levels of omentin in hemodialysis patients, diabetes mellitus, even under the enhancer effect of ESRD, led to lower levels of omentin in diabetic ESRD patients than in nondiabetic ESRD patients.

Unlike in previous studies no correlation was found between BMI (predialysis and postdialysis) and omentin levels of hemodialysis patients. This may be due to excessive levels of omentin (approximately 2-fold those in healthy subjects) in hemodialysis patients; therefore, BMI may have lost its statistical significance. Although there were overweight people in our study population, control and ESRD groups did not differ significantly in terms of BMI. Also, a significant correlation between waist circumference and omentin, shown in some studies [10, 11], was not found in our study but there was a negative non-significant trend between omentin and waist circumference ($r = -0.245$, $p = 0.06$). The underlying reason for this

may be the production of omentin especially by visceral adipose tissue rather than peripheral adipose tissue.

In conclusion, we found omentin levels to be elevated in hemodialysis patients with ESRD. In order to explain the significance of omentin levels in CKD, confirmatory studies are needed to evaluate the omentin levels in undialyzed uremic patients.

References

- 1 Foley RN, Parfrey PS, Sarnak MJ: Clinical epidemiology of cardiovascular disease in chronic renal disease. *Am J Kidney Dis* 1998; 32:S112–S119.
- 2 Vanholder R, Massy Z, Argiles A, Spasovski G, Verbeke F, Lameire N, European Uremic Toxin Work Group: Chronic kidney disease as cause of cardiovascular morbidity and mortality. *Nephrol Dial Transplant* 2005;20: 1048–1056.
- 3 Akdag I, Yilmaz Y, Kahvecioglu S, Bolca N, Ercan I, Ersoy A, Gullulu M: Clinical value of the malnutrition-inflammation-atherosclerosis syndrome for long-term prediction of cardiovascular mortality in patients with end-stage renal disease: a 5-year prospective study. *Nephron Clin Pract* 2008;108:C99–C105.
- 4 Coskun A, Bicik Z, Duran S, Alcelik A, Soypacaci Z, Yavuz O, Oksuz S: Pregnancy-associated plasma protein a in dialysis patients. *Clin Chem Lab Med* 2007;45:63–66.
- 5 Shoji T, Masakane I, Watanabe Y, Iseki K, Tsubakihara Y, Therapy JSD: Elevated non-high-density lipoprotein cholesterol (non-HDL-C) predicts atherosclerotic cardiovascular events in hemodialysis patients. *Clin J Am Soc Nephrol* 2011;6:1112–1120.
- 6 Tripepi G, Mattace Raso F, Sijbrands E, Seck MS, Maas R, Boger R, Witteman J, Rapisarda F, Malatino L, Mallamaci F, Zoccali C: Inflammation and asymmetric dimethylarginine for predicting death and cardiovascular events in ESRD patients. *Clin J Am Soc Nephrol* 2011;6:1714–1721.
- 7 Assadi M, Salimpour H, Akbarzadeh S, Nemati R, Jafari SM, Bargahi A, Samani Z, Seyedabadi M, Sanjdideh Z, Nabipour I: Correlation of circulating omentin-1 with bone mineral density in multiple sclerosis: the crosstalk between bone and adipose tissue. *PLoS One* 2011;6:e24240.
- 8 Moreno-Navarrete JM, Ortega F, Castro A, Sabater M, Ricart W, Fernandez-Real JM: Circulating omentin as a novel biomarker of endothelial dysfunction. *Obesity* 2011;19: 1552–1559.
- 9 Tan BK, Adya R, Randeve HS: Omentin: a novel link between inflammation, diabetes, and cardiovascular disease. *Trends Cardiovasc Med* 2010;20:143–148.
- 10 Liu R, Wang XL, Bu PL: Omentin-1 is associated with carotid atherosclerosis in patients with metabolic syndrome. *Diabetes Res Clin Pract* 2011;93:21–25.
- 11 Moreno-Navarrete JM, Catalan V, Ortega F, Gomez-Ambrosi J, Ricart W, Fruhbeck G, Fernandez-Real JM: Circulating omentin concentration increases after weight loss. *Nutr Metab (Lond)* 2010;7:27.
- 12 Tan BK, Adya R, Farhatullah S, Chen J, Lehnert H, Randeve HS: Metformin treatment may increase omentin-1 levels in women with polycystic ovary syndrome. *Diabetes* 2010;59:3023–3031.
- 13 Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC: Homeostasis model assessment – insulin resistance and beta-cell function from fasting plasma-glucose and insulin concentrations in man. *Diabetologia* 1985;28:412–419.
- 14 Mathur S, Devaraj S, Jialal I: Accelerated atherosclerosis, dyslipidemia, and oxidative stress in end-stage renal disease. *Curr Opin Nephrol Hypertens* 2002;11:141–147.
- 15 Oberg BP, McMenamin E, Lucas FL, McMonagle E, Morrow J, Ikizler TA, Himmel-farb J: Increased prevalence of oxidant stress and inflammation in patients with moderate to severe chronic kidney disease. *Kidney Int* 2004;65:1009–1016.
- 16 Shinohara K, Shoji T, Emoto M, Tahara H, Koyama H, Ishimura E, Miki T, Tabata T, Nishizawa Y: Insulin resistance as an independent predictor of cardiovascular mortality in patients with end-stage renal disease. *J Am Soc Nephrol* 2002;13:1894–1900.
- 17 Zanetti M, Barazzoni R, Guarnieri G: Inflammation and insulin resistance in uremia. *J Renal Nutr* 2008;18:70–75.
- 18 Yang RZ, Lee MJ, Hu H, Pray J, Wu HB, Hansen BC, Shuldiner AR, Fried SK, McLenithan JC, Gong DW: Identification of omentin as a novel depot-specific adipokine in human adipose tissue: possible role in modulating insulin action. *Am J Physiol Endocrinol Metab* 2006;290:E1253–E1261.
- 19 Lago F, Dieguez C, Gomez-Reino J, Gualillo O: Adipokines as emerging mediators of immune response and inflammation. *Nat Clin Pract Rheum* 2007;3:716–724.
- 20 Abdallah E, Waked E, Nabil M, El-Bendary O: Adiponectin and cardiovascular outcomes among hemodialysis patients. *Kidney Blood Press Res* 2012;35:247–253.
- 21 Axelsson J, Stenvinkel P: Role of fat mass and adipokines in chronic kidney disease. *Curr Opin Nephrol Hypertens* 2008;17:25–31.
- 22 Malyszko J, Malyszko JS, Mysliwiec M: Viscerofat and endothelial function in dialyzed patients. *Nephrology* 2010;15:190–196.
- 23 Rao M, Li LJ, Tighiouart H, Jaber BL, Pereira BJG, Balakrishnan VS, HEMO Study Group: Plasma adiponectin levels and clinical outcomes among haemodialysis patients. *Nephrol Dial Transplant* 2008;23:2619–2628.
- 24 Taskapan MC, Taskapan H, Sahin I, Keskin L, Atmaca H, Ozyalin F: Serum leptin, resistin, and lipid levels in patients with end stage renal failure with regard to dialysis modality. *Ren Fail* 2007;29:147–154.
- 25 Chudek J, Adamczak M, Karkoszka H, Budzinski G, Ignacy W, Funahashi T, Matsuzawa Y, Cierpka L, Kokot F, Wiecek A: Plasma adiponectin concentration before and after successful kidney transplantation. *Transplant Proc* 2003;35:2186–2189.
- 26 Huang JW, Yen CJ, Chiang HW, Hung KY, Tsai TJ, Wu KD: Adiponectin in peritoneal dialysis patients: a comparison with hemodialysis patients and subjects with normal renal function. *Am J Kidney Dis* 2004;43: 1047–1055.
- 27 Tsao YT, Hsu YJ, Chu NF, Lai CH, Chiu JS, Lin SH: Association of plasma adiponectin and cardiovascular risk profiles in nondiabetic uremic patients on peritoneal dialysis. *J Nephrol* 2008;21:744–752.
- 28 Brunzell JD, Hokanson JE: Dyslipidemia of central obesity and insulin resistance. *Diabetes Care* 1999;22(suppl 3):C10–C13.
- 29 de Souza Batista CM, Yang RZ, Lee MJ, Glynn NM, Yu DZ, Pray J, Ndubuizu K, Patil S, Schwartz A, Kligman M, Fried SK, Gong DW, Shuldiner AR, Pollin TI, McLenithan JC: Omentin plasma levels and gene expression are decreased in obesity. *Diabetes* 2007; 56:1655–1661.
- 30 Tan BK, Pua S, Syed F, Lewandowski KC, O'Hare JP, Randeve HS: Decreased plasma omentin-1 levels in type 1 diabetes mellitus. *Diabet Med* 2008;25:1254–1255.
- 31 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;88:29–33.