Omentin-1 Levels and Outcomes in Incident Peritoneal Dialysis Patients

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Rationale & Objective: Omentin-1 is an adipokine with anti-inflammatory and cardioprotective properties. The objective of this study was to determine the prognostic role of plasma omentin-1 levels in incident peritoneal dialysis (PD) patients.

Study Design: Retrospective analysis of prospective cohort.

Setting & Participants: 152 incident PD patients.

Predictors: Plasma omentin-1 level, adipose tissue omentin-1 messenger RNA (mRNA) expression.

Outcomes: Patient survival, technique survival, hospital admission, and duration of stay.

Analytical Approach: Time-to-event survival analyses; linear regression for hospitalization.

Results: The mean age was 58.4 ± 11.7 years; 102 were men, and 92 had diabetes. There was no significant correlation between plasma omentin-1 level and its adipose tissue mRNA expression. A higher plasma omentin-1 level quartile was not associated with patient survival ($P = 0.92$) or technique survival ($P = 0.83$) but had a modest correlation with a lower number of hospital admissions ($P = 0.07$) and shorter duration of hospital stay ($P = 0.04$). In adjusted models using multivariable linear regression, a higher plasma omentin-1 level quartile remained significantly associated with fewer hospital admissions (β, -0.13; 95% CI, -0.26 to -0.002; $P = 0.05$) and shorter hospitalization duration (β, -0.20; 95% CI, -0.38 to -0.02; $P = 0.03$).

Limitations: Observational study with baseline measures only.

Conclusions: Plasma omentin-1 level was not associated with patient survival, technique survival, or peritonitis, but higher plasma omentin-1 levels were associated with fewer hospital admissions and shorter duration of hospitalization among incident PD patients.

Peritoneal dialysis (PD) is a life-saving kidney replacement therapy.[1,2] With the advances in dialysis technology and reduction in peritonitis rate, cardiovascular disease has become the major cause of mortality and morbidity in PD patients.[3] In addition to the high prevalence of traditional cardiovascular risk factors, the role of non-traditional cardiovascular disease risk factor in dialysis patients has been increasingly recognized.[4,5]

Obesity is common in the general population[6,7] as well as among kidney failure patients newly started on PD.[8] There is a wealth of literature to support that obesity is a risk factor for technique failure and probably mortality in PD.[9,10] In the past 2 decades, it has been recognized that adipose tissue is an active endocrine and metabolic organ that contributes to the systemic inflammatory state by producing various adipokines with distant effects,[11,12] and omentin-1 is a notable adipokine in this regard.

Omentin-1 is an insulin-sensitizing adipokine with anti-inflammatory, anti-atherosclerotic, and cardioprotective properties.[13,14] Plasma omentin-1 level is decreased in obesity but elevated in chronic kidney disease.[15-18] Plasma omentin-1 level was inversely correlated with carotid intima-media thickness in patients with type 2 diabetes and high plasma adiponectin levels,[19] and low serum omentin-1 level was associated with poor prognosis in heart failure.[20] However, previous studies examined plasma omentin-1 level, which represents the omentin-1 secretion capacity of each adipocyte in combination with the total body adipose tissue mass. In the present study, we examined the prognostic roles of plasma omentin-1 levels and its corresponding mRNA expression in adipose tissue in incident PD patients.

METHODS

Overall Design

This is a retrospective analysis of a prospective observational study, which was approved by the Joint Chinese University of Hong Kong – New Territories East Cluster Clinical Research Ethics Committee (approval number CREC-2008.554). All study procedures were in compliance with the Declaration of Helsinki. We recruited 152 consecutive adult incident PD patients in our center. After written informed consent, 1 to 2 grams of subcutaneous and preperitoneal adipose tissue samples were obtained during mini-laparotomy for PD catheter insertion. The subcutaneous and preperitoneal adipose tissue of 6 patients without kidney disease but who required abdominal surgery were used as the healthy control group. Four to 6 weeks after the patients were stable on PD, we performed routine biochemical tests, peritoneal transport study, anthropometric measurement, dialysis adequacy and nutritional

PLAIN-LANGUAGE SUMMARY

Omentin-1 is an adipokine with anti-inflammatory and cardioprotective properties. The objective of this study was to determine the prognostic role of plasma omentin-1 levels in a cohort of 152 incident peritoneal dialysis (PD) patients. Although a higher plasma omentin-1 level quartile did not predict patient survival or technique survival, it was significantly associated with a lower number of hospital admissions and shorter duration of hospital stay in the subsequent 2 years. In contrast, adipose tissue omentin-1 mRNA expression level or PD effluent omentin-1 level was not associated with clinical outcomes. Our results suggest that plasma omentin-1 level may be a valuable tool for risk stratification of PD patients.

status assessment, multifrequency bioimpedance spectroscopy study, and arterial pulse wave velocity study. Plasma and peritoneal dialysis effluent samples were collected at the same time for omentin-1 level measurement. Charlson's comorbidity index was computed as previously described.[21]

Adipose Tissue Omentin-1 mRNA Expression

All adipose tissue specimens were processed immediately and stored at -80°C. Total RNA was extracted from the adipose tissue specimen using the miRNeasy Mini Kit (Qiagen). Reverse transcription was performed using the Invitrogen cDNA Synthesis Kit and SuperScript IV VILO Master Mix (both from ThermoFisher Scientific) according to the manufacturer's instructions. Omentin-1 mRNA expression in the adipose tissue was measured by real-time quantitative polymerase chain reaction, using the Applied Biosystems Step One Plus system. Commercially available TaqMan primers and probes, including 2 unlabeled polymerase chain reaction primers and 1 fluorescein amidites dye-labeled TaqMan minor groove binder probe, were used (all from Applied Biosystems). Phosphoglycerate kinase-1 was used as the housekeeping gene because of its stable expression in human adipose tissue.[22] The expression of omentin-1 mRNA was compared to that of the adipose tissue of 6 individuals with no kidney disease. All samples were performed in triplicate. The results were analyzed with the Sequence Detection Software v2.0 (Applied Biosystems). The ΔΔCT method for relative quantitation was used.[23]

Plasma and PDE Omentin-1 Levels

Plasma and PDE omentin-1 levels were measured using a commercially available kit (Omentin-1 Human ELISA Kit, BioVendor) following the manufacturer's instructions. All assays were performed in duplicate. The detection limit of omentin-1 was 0.5 ng/mL; the interassay coefficient of variation was 4.6%.

Study of Peritoneal Transport

The standard peritoneal permeability test was performed 4 to 6 weeks after the patients were stable on PD by the method of Twardowski and has been described previously.[24] In enzyme-linked immunosorbent assaythis study, dialysate-to-plasma ratios of creatinine at 4 hours were calculated after correction for glucose interference. Mass transfer area coefficients of creatinine normalized for body surface area were calculated using a standard formula.[25] Body surface area was determined by the formula of Gehan and George.[26]

Anthropometric Measurements

On the same day of the peritoneal permeability test, we also performed standard anthropometric measurements, including body weight, body mass index, waist circumference, hip circumference, mid-arm circumference, triceps, and subscapular skinfold thickness. The waist-hip ratio was also computed.

Dialysis Adequacy and Nutritional Status

The method of dialysis adequacy assessment has been described previously.[27] In essence, 24-hour urine and dialysate collection were performed for the calculation of the total Kt/V. Residual kidney function was represented by the residual glomerular filtration rate, which was calculated as the average of 24-hour urinary urea and creatinine clearances.[28] Nutritional status was represented by serum albumin level, subjective global assessment score, comprehensive malnutrition-inflammation score, normalized protein nitrogen appearance, and fat-free edema-free body mass. For subjective global assessment, the 4-item 7-point scoring system validated in PD patients was used.[29] The calculation of malnutrition-inflammation score included 10 items, each scored from 0 to 3, with a total score of 30.[30] Normalized protein nitrogen appearance was calculated by the modified Bergstrom's formula.[31] Fat-free edema-free body mass was determined by the creatinine kinetic method according to the formula described by Forbes and Bruining[32] and presented as the percentage of ideal body weight.

Multifrequency bioimpedance spectroscopy study

We used the multifrequency device (Body Composition Monitor, Fresenius Medical Care) as described previously.[33,34] In this study, we analyzed the data on lean tissue mass, adipose tissue mass, the volume of overhydration, and extracellular-to-intracellular volume ratio.

Arterial Pulse Wave Velocity Study

Arterial pulse wave velocity was measured by an automatic computerized recorder and analyzed using the Complior SP program (Artech Medical) by the method described previously.[35] In the present report, we

computed the carotid-radial and carotid-femoral pulse wave velocity.

Clinical Outcome

After the initial assessment, all patients were followed for up to 5 years. During the follow-up period, the clinical management was decided by individual clinicians and not affected by the study. The primary outcomes were patient survival and technique survival. For patient survival, recovery of kidney function, loss to follow-up, and transfer to other dialysis centers were censored, while conversion to long-term hemodialysis and kidney transplant were treated as competing events. For the analysis of technique survival, patient death and kidney transplant were treated as competing events, while recovery of kidney function, loss to follow-up, and transfer to other dialysis centers were censored. Secondary outcome measures of this study included the number of hospital admission and duration of hospital stay (both adjusted for the duration of follow-up and computed for each individual patient), peritonitis rate, and the rate of residual kidney function decline during the first 2 years of PD.

Statistical Analysis

Statistical analysis was performed by SPSS for Windows software version 25.0 (IBM). The normality of data distribution was checked by the Shapiro-Wilk Test. Summary statistics were described as frequency (percentage) for categorical variables and mean ± standard deviation or median (interquartile range [IQR]) for continuous variables, as appropriate. Patients were grouped into plasma omentin-1 level quartiles for comparison to explore the correlation between omentin-1 level and other clinical parameters. Demographic and clinical data were compared between quartiles of plasma omentin-1 level by χ^2 test for categorical variables and Jonckheere-Terpstra modification of the Kruskal-Wallis test for continuous or ordinal variables. Correlation between variables was explored by Spearman's rank correlation coefficient. Survival rates were analyzed by the Kaplan-Meier survival curves and compared by the log-rank test between plasma omentin-1 level quartiles. For the rate of hospital admission and duration of hospital stay, data were first compared between plasma omentin-1 level quartiles by the Jonckheere-Terpstra test. Since the result of univariate analysis for the hospitalization data showed marginal significance, multiple linear regression models were conducted after log-transformation of the hospitalization as previously described.[36] In addition to the plasma omentin-1 level quartile, Charlson's comorbidity score, serum albumin, mass transfer area coefficients of creatinine, total weekly Kt/V, residual glomerular filtration rate, normalized protein nitrogen appearance, fat-free edema-

Table 1. Baseline Demographic and Clinical Characteristics According to the Plasma Omentin-1 Level Quartile

		Plasma Omentin-1 Level Quartile				
	All Cases	**I**	**II**	**III**	**IV**	**P**
No. of patients	152	38	38	38	38	
Plasma omentin-1 level, ng/mL	940.1 ± 351.0	551.1 ± 124.3	802.9 ± 57.7	988.5 ± 71.1	1417.7 ± 260.1	<0.001[a]
Age, y	58.4 ± 11.7	58.1 ± 12.2	58.4 ± 12.2	58.0 ± 11.8	59.1 ± 11.0	0.95[a]
Sex, n (%)						0.57[b]
Male	102 (67.1%)	26 (68.4%)	23 (60.5%)	26 (68.4%)	27 (71.1%)	
Female	50 (32.9%)	12 (31.6%)	15 (39.5%)	12 (31.6%)	11 (28.9%)	
Blood pressure, mm Hg						
Systolic	140 ± 19	140 ± 21	143 ± 14	137 ± 18	142 ± 20	0.71[a]
Diastolic	76 ± 13	78 ± 14	77 ± 12	73 ± 11	75 ± 14	0.61[a]
Primary kidney disease, n (%)						0.92[b]
Diabetes mellitus	76 (50.0%)	19 (50.0%)	15 (39.5%)	21 (55.3%)	21 (55.3%)	
Hypertension	14 (9.2%)	5 (13.2%)	5 (13.2%)	2 (5.3%)	2 (5.3%)	
Glomerulonephritis	31 (20.4%)	7 (18.4%)	9 (23.7%)	6 (15.8%)	9 (23.7%)	
Polycystic kidney disease	3 (2.0%)	1 (2.6%)	1 (2.6%)	1 (2.6%)	0 (0.0%)	
Urological	6 (3.9%)	2 (5.3%)	2 (5.3%)	1 (2.6%)	1 (2.6%)	
Others	2 (1.3%)	1 (2.6%)	0 (0.0%)	1 (2.6%)	0 (0.0%)	
Unknown	20 (13.1%)	3 (7.9%)	6 (15.8%)	6 (15.8%)	5 (13.2%)	
Major comorbidities, n (%)						
Diabetes mellitus	92 (60.5%)	22 (57.9%)	22 (57.9%)	24 (63.2%)	24 (63.2%)	0.42[b]
Ischemic heart disease	40 (26.3%)	12 (31.6%)	9 (23.7%)	8 (21.1%)	11 (28.9%)	0.81[b]
Cerebrovascular accident	28 (18.4%)	8 (21.1%)	6 (15.8%)	8 (21.1%)	6 (15.8%)	0.76[b]
Peripheral vascular disease	12 (7.9%)	2 (5.3%)	2 (5.3%)	4 (10.5%)	4 (10.5%)	0.27[b]
Charlson's comorbidity score	6.2 ± 2.5	6.08 ± 2.27	6.06 ± 2.91	6.08 ± 2.39	6.39 ± 2.51	0.67[a]

Data are compared by [a]Jonckheere-Terpstra test and [b]χ^2 test for linearity trend.

Table 2. Baseline Anthropometric and Biochemical Characteristics According to the Plasma Omentin-1 Level Quartile

| | Plasma Omentin-1 Level Quartile | | | | | |
	All Cases	I	II	III	IV	P^a
No. of patients	152	38	38	38	38	
PDE omentin-1 level, ng/mL	10.17 ± 9.02	9.71 ± 9.42	9.08 ± 8.18	10.08 ± 8.55	11.85 ± 10.02	0.68
Adipose tissue omentin-1 mRNA[b]	0.67 (0.10-137.30)	1.77 (0.09-165.30)	3.65 (0.27-137.30)	0.58 (0.08-165.81)	0.45 (0.17-14.96)	0.52
Anthropometric measures						
Body weight, kg	66.7 ± 14.3	68.4 ± 16.4	67.1 ± 15.4	65.8 ± 11.2	63.3 ± 13.3	0.20
Body mass index, kg/m²	24.8 ± 4.2	25.8 ± 5.0	25.5 ± 4.1	24.5 ± 3.1	23.5 ± 3.9	0.02
Waist circumference, cm	88.8 ± 11.6	92.6 ± 12.5	88.4 ± 11.7	88.1 ± 10.4	86.1 ± 11.1	0.03
Hip circumference, cm	95.6 ± 9.1	97.8 ± 10.7	96.3 ± 10.2	94.2 ± 7.0	93.8 ± 7.9	0.05
Waist-hip ratio	0.93 ± 0.07	0.94 ± 0.06	0.92 ± 0.09	0.93 ± 0.07	0.92 ± 0.07	0.09
Mid-arm circumference, cm	25.9 ± 3.0	26.4 ± 3.7	26.7 ± 2.6	25.5 ± 2.4	25.0 ± 3.0	0.06
Triceps skin fold	9.8 ± 3.7	10.3 ± 4.1	10.5 ± 3.2	9.3 ± 3.7	9.0 ± 3.8	0.09
Subscapular skin fold	11.5 ± 4.8	11.7 ± 4.7	12.7 ± 4.6	10.5 ± 5.0	11.0 ± 4.7	0.19
Other nutritional scores						
SGA	5.31 ± 0.87	5.24 ± 1.00	5.15 ± 0.93	5.29 ± 0.83	5.55 ± 0.69	0.13
MIS	6.7 ± 3.6	8.0 ± 3.7	6.1 ± 4.0	6.9 ± 3.7	5.7 ± 2.7	0.07
Total Kt/V	2.05 ± 0.63	2.04 ± 0.72	2.23 ± 0.58	1.94 ± 0.60	2.02 ± 0.62	0.49
Residual GFR, mL/min/1.73 m²	3.96 ± 2.67	3.42 ± 3.10	4.95 ± 2.97	3.94 ± 2.06	3.54 ± 2.26	0.86
Peritoneal transport characteristics						
D/P4	0.69 ± 0.12	0.68 ± 0.13	0.69 ± 0.12	0.70 ± 0.12	0.68 ± 0.12	0.67
MTAC, mL/min/1.73 m²	10.91 ± 5.04	10.57 ± 5.70	11.42 ± 5.85	11.08 ± 4.55	10.57 ± 4.00	0.46
Hemoglobin, g/dL	8.94 ± 1.20	8.78 ± 1.14	9.35 ± 1.34	8.85 ± 1.29	8.83 ± 0.97	0.99
Albumin, g/L	35.28 ± 4.42	34.05 ± 4.27	35.91 ± 5.18	36.14 ± 3.89	35.12 ± 4.17	0.26
Serum lipid profile, mmol/L						
Total cholesterol	4.53 ± 1.21	4.52 ± 1.44	4.30 ± 0.93	4.66 ± 1.29	4.62 ± 1.12	0.24
Triglyceride	1.52 ± 0.86	1.60 ± 1.06	1.76 ± 0.83	1.52 ± 0.74	1.24 ± 0.71	0.04
LDL cholesterol	2.59 ± 1.04	2.63 ± 1.20	2.42 ± 0.78	2.70 ± 1.13	2.60 ± 1.00	0.63
HDL cholesterol	1.26 ± 0.39	1.16 ± 0.32	1.09 ± 0.33	1.28 ± 0.33	1.49 ± 0.45	<0.001
NPNA, g/kg/d	1.13 ± 0.24	1.10 ± 0.30	1.14 ± 0.22	1.11 ± 0.19	1.17 ± 0.25	0.49
FEBM, %	40.48% ± 13.05%	41.52% ± 12.10%	39.21% ± 17.10%	39.15% ± 10.92%	42.10% ± 11.70%	0.78
Bioimpedance spectroscopy						
Overhydration, L	4.53 ± 3.25	4.59 ± 3.37	4.51 ± 3.99	4.12 ± 2.80	4.92 ± 2.85	0.73
E:I ratio	1.01 ± 0.17	1.04 ± 0.19	1.01 ± 0.20	0.98 ± 0.15	1.02 ± 0.15	0.46
Lean tissue mass, kg	41.41 ± 10.98	40.81 ± 12.13	42.04 ± 10.16	41.37 ± 9.39	41.50 ± 12.36	0.77
Adipose tissue mass, kg	20.04 ± 11.13	21.66 ± 13.10	22.44 ± 12.47	19.21 ± 9.75	16.92 ± 8.10	0.09
Pulse wave velocity, cm/s						
Carotid-radial	10.51 ± 1.35	10.52 ± 1.08	10.52 ± 1.04	10.80 ± 1.76	10.20 ± 1.38	0.41

(Continued)

Table 2 (Cont'd). Baseline Anthropometric and Biochemical Characteristics According to the Plasma Omentin-1 Level Quartile

| | All Cases | Plasma Omentin-1 Level Quartile | | | | P^a |
		I	II	III	IV	
Carotid-femoral	11.44 ± 2.35	11.82 ± 2.31	11.14 ± 2.28	11.45 ± 2.48	11.31 ± 2.38	0.37
C-reactive protein, mg/L	12.96 ± 29.58	24.60 ± 41.48	8.79 ± 16.98	6.70 ± 15.15	10.95 ± 32.61	0.003
C peptide, ng/mL	9.10 ± 7.09	11.19 ± 8.34	9.66 ± 8.29	8.30 ± 6.98	7.18 ± 2.99	0.10

Abbreviations: D/P4, dialysate-to-plasma creatinine concentration at 4 h; E:I ratio, extracellular-to-intracellular volume ratio; FEBM, fat-free edema-free body mass; GFR, glomerular filtration rate; HDL, high-density lipoprotein; LDL, low-density lipoprotein; MIS, malnutrition-inflammation score; MTAC, mass transfer areas coefficient of creatinine; NPNA, normalized protein nitrogen appearance; PDE, peritoneal dialysis effluent; SGA, subjective global assessment overall score.
[a]Data are compared by the Jonckheere-Terpstra test. [b]Data presented as median (interquartile range).

free body mass, carotid-femoral pulse wave velocity, overhydration volume, serum C-reactive protein, and total cholesterol level were included for the construction of the multiple linear regression models for hospitalization data. These parameters were included because of their reported prognostic role in PD patients. A P value of < 0.05 was considered statistically significant. All probabilities were 2-tailed.

RESULTS

We recruited 152 new PD patients. Their average age was 58.4 ± 11.7 years; 102 patients (67.1%) were men, and 92 (60.5%) had diabetes. The median baseline plasma omentin-1 level was 879.9 (IQR, 691.5-1136.5) ng/mL, while PDE omentin-1 level was 7.48 (IQR, 3.35-14.53) ng/mL. The corresponding median adipose tissue omentin-1 mRNA expression was 0.67 (IQR, 0.10-137.30) fold of the healthy control group. The baseline demographic and clinical characteristics of patients are grouped according to the plasma omentin-1 level quartile and summarized in Table 1 and Table 2, respectively. In essence, there were significant differences in body mass index, waist circumference, serum triglyceride, high-density lipoprotein cholesterol, and serum C-reactive protein level between plasma omentin-1 level quartiles. Post hoc analysis showed significant differences in body mass index and waist circumference between omentin-1 level quartiles I and IV. Serum triglyceride levels of omentin-1 level quartile IV were significantly lower, and high-density lipoprotein cholesterol level higher, than all other quartile groups, while serum C-reactive protein level of omentin-1 level quartile I was significantly higher than all other quartile groups.

Relation with Baseline Biochemical Measures

There was no significant correlation between plasma omentin-1 level with adipose tissue omentin-1 mRNA expression (r = -0.034, P = 0.69) or PDE omentin-1 level (r = 0.082, P = 0.37). Adipose tissue omentin-1 mRNA expression also did not correlate with PDE omentin-1 level (r = 0.063, P = 0.49). The relation between plasma, PDE, adipose tissue omentin-1 levels, and other clinical parameters are summarized in Table 3. There were moderate but statistically significant correlations between plasma and adipose tissue omentin-1 levels with various anthropometric measurements (Table 3). Plasma omentin-1 level also showed modest but significant correlation with baseline C-reactive protein (r = -0.26, P = 0.002) and serum C-peptide level (r = -0.19, P = 0.02). In contrast, PDE omentin-1 level significantly correlated with peritoneal transport parameters, including dialysate-to-plasma ratios of creatinine at 4 hours and mass transfer area coefficients of creatinine (Fig 1), but not with other anthropometric measurements.

Patient and Technique Survival

The average follow-up period was 46.8 ± 31.9 months. During the follow-up period, 90 patients (59.2%) died,

Table 3. Correlation Between Omentin-1 Level and Baseline Clinical and Biochemical Parameters

| Parameters	Omentin-1 level		
	Plasma Level	Adipose Tissue mRNA	PDE Level
Charlson's score	r = 0.010, P = 0.93	r = 0.03, P = 0.75	r = -0.08, P = 0.38
Body weight	r = -0.13, P = 0.11	r = -0.00, P = 0.02	r = 0.10, P = 0.28
Body mass index	r = - 0.22, P = 0.01	r = - 0.06, P = 0.47	r = 0.01, P = 0.94
Waist circumference	r = - 0.18, P = 0.02	r = - 0.21, P = 0.01	r = 0.03, P = 0.72
Hip circumference	r = -0.18, P = 0.03	r = -0.19, P = 0.02	r = 0.05, P = 0.61
Waist-hip ratio	r = -0.17, P = 0.05	r = -0.19, P = 0.02	r = -0.04, P = 0.70
Mid-arm circumference	r = -0.17, P = 0.05	r = -0.12, P = 0.15	r = -0.03, P = 0.71
Triceps skin fold	r = -0.14, P = 0.09	r = 0.11, P = 0.18	r = -0.07, P = 0.47
Subscapular skin fold	r = -0.12, P = 0.16	r = 0.02, P = 0.79	r = -0.01, P = 0.90
SGA	r = 0.18, P = 0.13	r = -0.11, P = 0.37	r = -0.07, P = 0.56
MIS	r = -0.24, P = 0.04	r = 0.06, P = 0.64	r = 0.14, P = 0.25
Total Kt/V	r = -0.05, P = 0.61	r = 0.27, P = 0.002	r = -0.03, P = 0.74
Residual GFR	r = -0.00, P = 0.99	r = 0.09, P = 0.32	r = -0.06, P = 0.55
D/P4	r = 0.01, P = 0.91	r = 0.03, P = 0.69	r = 0.50, P < 0.001
MTAC	r = 0.04, P = 0.62	r = 0.09, P = 0.31	r = 0.42, P < 0.001
Hemoglobin	r = -0.01, P = 0.96	r = -0.13, P = 0.13	r = -0.09, P = 0.35
Albumin	r = 0.13, P = 0.13	r = 0.07, P = 0.43	r = -0.30, P = 0.001
Total cholesterol	r = 0.08, P = 0.36	r = -0.02, P = 0.86	r = 0.23, P = 0.01
Triglycerides	r = -0.15, P = 0.07	r = 0.07, P = 0.39	r = -0.023, P = 0.76
LDL cholesterol	r = 0.03, P = 0.76	r = -0.09, P = 0.27	r = 0.15, P = 0.09
HDL cholesterol	r = 0.32, P < 0.001	r = 0.04, P = 0.61	r = 0.23, P = 0.01
NPNA	r = 0.07, P = 0.43	r = -0.06, P = 0.53	r = -0.00, P = 0.96
FEBM	r = 0.04, P = 0.66	r = -0.15, P = 0.09	r = 0.04, P = 0.65
Overhydration	r = -0.00, P = 0.98	r = -0.04, P = 0.63	r = 0.27, P = 0.003
E:I ratio	r = -0.08, P = 0.37	r = 0.06, P = 0.46	r = 0.09, P = 0.30
Lean tissue mass	r = 0.01, P = 0.87	r = -0.18, P = 0.03	r = 0.17, P = 0.06
Adipose tissue mass	r = -0.15, P = 0.09	r = 0.00, P = 0.96	r = -0.16, P = 0.08
Carotid-radial PWV	r = -0.08, P = 0.38	r = 0.09, P = 0.28	r = 0.03, P = 0.73
Carotid-femoral PWV	r = -0.06, P = 0.50	r = 0.21, P = 0.02	r = 0.05, P = 0.63
C-reactive protein	r = -0.26, P = 0.002	r = 0.03, P = 0.74	r = -0.10, P = 0.31
C-peptide level	r = -0.19, P = 0.02	r = -0.13, P = 0.12	r = -0.03, P = 0.71

Abbreviations: D/P4, dialysate-to-plasma creatinine concentration at 4 h; E:I ratio, extracellular-to-intracellular volume ratio; FEBM, fat-free edema-free body mass; GFR, glomerular filtration rate; HDL, high-density lipoprotein; LDL, low-density lipoprotein; MIS, malnutrition-inflammation score; MTAC, mass transfer areas coefficient of creatinine; NPNA, normalized protein nitrogen appearance; PDE, peritoneal dialysis effluent; PWV, pulse wave velocity; SGA, subjective global assessment overall score.

17 patients (11.2%) were switched to long-term hemodialysis, 15 patients (9.9%) had a kidney transplant, and 6 patients (3.9%) were transferred to other centers. The causes of death were ischemic heart diseases (23 patients, 15.1%), cerebrovascular accidents (9 patients, 5.9%), peritonitis (10 patients, 6.6%), non-peritonitis infection (34 patients, 22.4%), malignancy (3 patients, 2.0%), termination of dialysis (2 patients, 1.3%), sudden death (5 patients, 3.3%), and other specific causes (4 patients, 2.6%). The Kaplan-Meier plots of patient and technique survival are summarized in Fig 2. The 5-year patient survival rates of plasma omentin-1 level quartiles, from lowest to highest, were 41.0%, 38.9%, 41.6%, and 46.6%, respectively (log-rank test, P = 0.92), and the corresponding technique survival rates were 85.7%, 85.2%, 78.4%, and 85.7%, respectively (P = 0.83). Similarly, the 5-year patient survival rates of adipose tissue omentin-1 mRNA expression

quartiles, from the lowest to highest, were 40.2%, 51.7%, 43.3%, and 33.7% respectively (P = 0.91), and the corresponding technique survival rates were 88.9%, 83.7%, 84.8%, and 84.2%, respectively (P = 0.81). There was also no relation between PDE omentin-1 level and patient or technique survival. There was no significant interaction between the degree of adiposity (body mass index or waist circumference) and omentin-1 level quartiles on patient or technique survival.

Relation With Hospitalization

During the first 2 years of follow-up, there were 862 hospital admissions for a total of 6,082 days. The median hospital admission rate was 1.59 (IQR, 0.84-3.27) episodes per year, and the median duration of hospital stay was 9.53 (IQR, 2.96-21.05) days per year. There were modest differences in the number of hospital admission
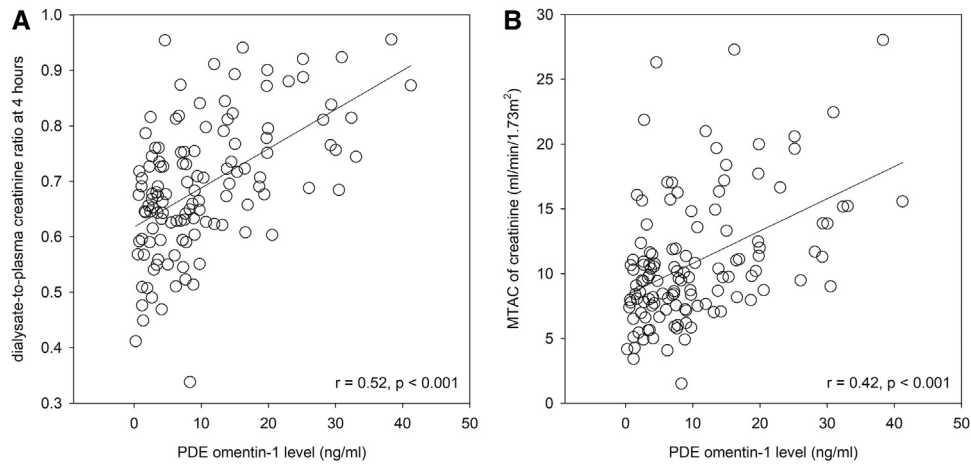
Figure 1. Correlation between peritoneal dialysis effluent (PDE) omentin-1 level with peritoneal transport characteristics: (A) dialysate-to plasma ratio of creatinine at 4 h; and (B) mass transfer area coefficient (MTAC) of creatinine. The Spearman's rank correlation coefficient is depicted.

and duration of hospital stay between plasma omentin-1 level quartiles, although the latter did not reach statistical significance (Fig 3). Post hoc analysis showed significant differences in hospital admission rate between omentin-1 level quartile I and IV. With multivariable linear regression analyses adjusting confounders, the plasma omentin-1 level quartile remained a significant predictor of the rate of hospital admission as well as duration of hospital stay
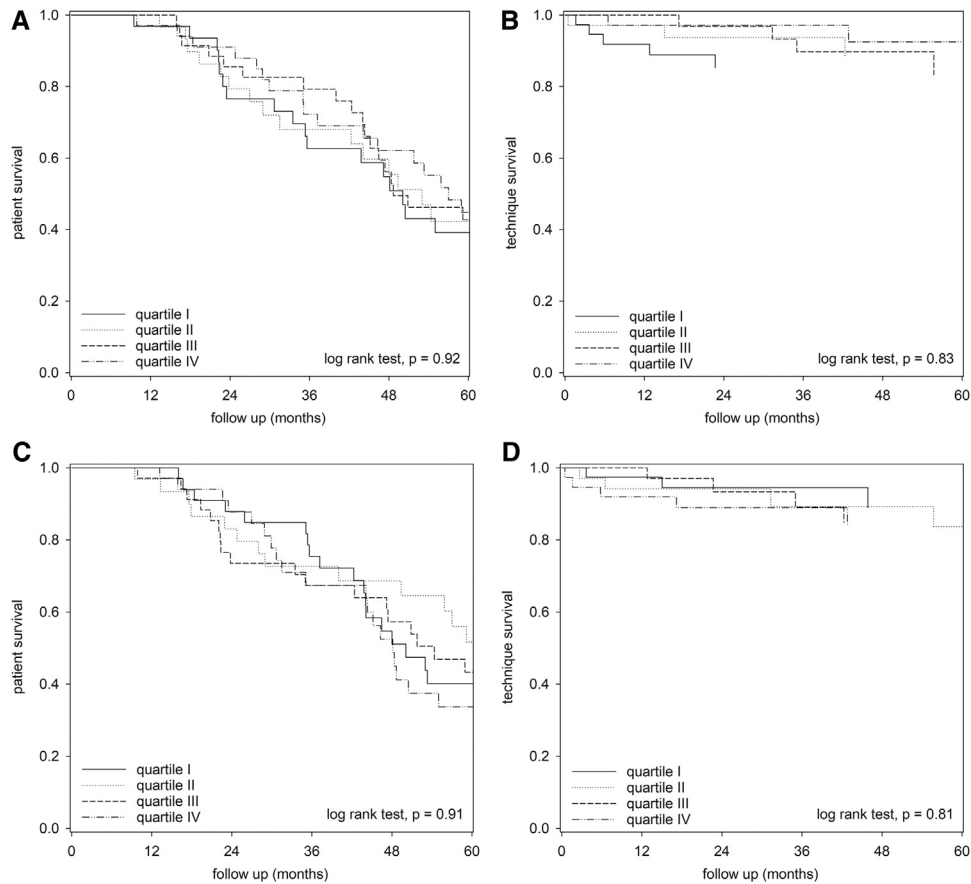
Figure 2. Kaplan-Meier plot on the effect of plasma omentin-1 level quartile on (A) patient survival; and (B) technique survival; and adipose tissue omentin-1 mRNA level quartile on (C) patient survival; and (D) technique survival. Quartile I denotes the lowest level, whereas quartile IV denotes the highest.
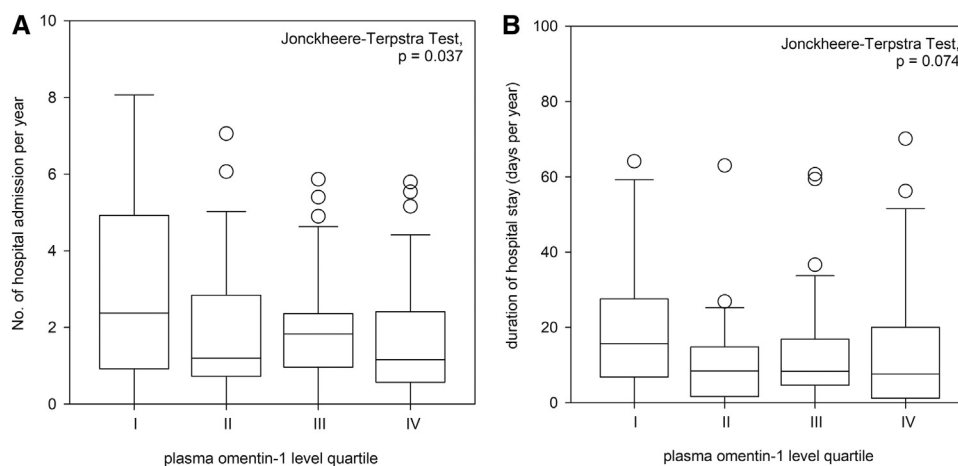
Figure 3. The effect of plasma omentin-1 level quartile on (A) number of hospital admission; and (B) duration of hospital stay. Quartile I denotes the lowest level, whereas quartile IV denotes the highest plasma omentin-1 level.

(Table 4). In contrast, there was no significant correlation between the rate of hospital admission or duration of hospitalization with adipose tissue omentin-1 expression or with PDE omentin-1 level.

Other Secondary Outcomes

During the first 2 years of follow-up, 63 patients (41.4%) developed anuria. The median rate of residual kidney function decline was -1.22 (IQR, -2.22 to -0.53) mL/min/1.73 m^2 per year. There was no significant correlation between rate of residual glomerular filtration rate decline and plasma omentin-1 level (r = 0.09, P = 0.35), its adipose tissue mRNA expression (r = 0.06, P = 0.51), or PDE omentin-1 level (r = -0.003, P = 0.98). There was also no significant association between the time to anuria and plasma or PDE omentin-1 level, or its adipose tissue mRNA level.

During the first 2 years of follow-up, 89 patients (58.6%) had 188 peritonitis episodes. The overall peritonitis rate was 0.77 episodes per patient-year. There was no significant correlation between plasma or PDE omentin-1 level or its adipose tissue mRNA level and the peritonitis rate.

DISCUSSION

In the present study, we found that a higher plasma omentin-1 level quartile had a modest but significant association with a lower number of subsequent hospital admissions and shorter duration of hospital stay. In contrast, the plasma omentin-1 level quartile did not predict patient or technique survival of incident PD patients.

Unlike previous reports, our present study focused on PD patients, and we performed a comprehensive assessment by measuring omentin-1, including plasma and PDE levels as well as its adipocyte mRNA expression. The absolute plasma omentin-1 level in our patients was similar

to that reported in regular hemodialysis patients but much higher than normal healthy controls.[18] Like other peptide mediators, omentin-1 is partially degraded by the kidney, and patients with kidney failure are expected to have an elevated plasma level. Moreover, malnutrition is common in dialysis patients,[38] which also contributes to a higher plasma omentin-1 level.[18,39,40]

The results of our present study is complementary to our previous report on plasma and adipose tissue adiponectin level in incident PD patients,[41] which was derived from the same cohort of patients. Our previous study found that plasma adiponectin level significantly correlated with the degree of adiposity but was not associated with the clinical outcomes of incident PD patients,[41] whereas the present study showed modest correlation between plasma omentin-1 level and adiposity as well as hospitalization. Our results indicate that each adipokine has distinct clinical relevance, and plasma omentin-1 level appears to be a superior biomarker in incident PD patients.

In our present study, there was no association between plasma omentin-1 level and arterial pulse wave velocity. Our findings are in line with the previous report of Sengul et al,[16] which found no correlation between plasma omentin-1 level and carotid intima-media thickness in non-diabetic chronic kidney disease patients. In contrast, Kocijancic et al[42] found a significant inverse correlation between plasma omentin-1 level and carotid intima-media thickness, and the omentin-1 level was also a strong predictor of cardiovascular death in hemodialysis patients with subclinical atherosclerosis. However, the actual correlation coefficient was modest, with only 6% of the variation in carotid intima-media thickness being related to the omentin-1 level.[42] Another study from the same group also found that plasma omentin-1 levels in hemodialysis patients with diabetes were lower than in hemodialysis patients without diabetes,[43] which was not observed in our study. Because of the limitations in the original study

Table 4. Multivariable Linear Regression for Hospitalization[a]

Variable	β	95% CI	P
Number of Hospital Admission			
(Constant)	-0.05	-2.36 to 2.25	0.96
Plasma omentin-1 level quartile	-0.13	-0.26 to -0.002	0.05
CCI	0.11	0.04 to 0.18	0.003
Serum albumin	-0.01	-0.05 to 0.03	0.76
MTAC	0.02	-0.01 to 0.06	0.13
Total Kt/V	0.35	0.002 to 0.69	0.05
Residual GFR	-0.11	-0.22 to -0.01	0.04
NPNA	0.20	-0.55 to 0.94	0.60
FEBM	-0.01	-0.03 to 0.01	0.26
CF-PWV	-0.04	-0.11 to 0.03	0.28
Overhydration	0.04	-0.01 to 0.09	0.09
Serum CRP	-0.01	-0.01 to 0.002	0.13
Total cholesterol	0.10	-0.02 to 0.23	0.10
Duration of Hospitalization			
(Constant)	2.42	-0.72 to 5.57	0.13
Plasma omentin-1 level quartile	-0.20	-0.38 to -0.02	0.03
CCI	0.16	0.07 to 0.26	0.001
Serum albumin	-0.04	-0.09 to 0.02	0.17
MTAC	0.04	0.001 to 0.09	0.05
Total Kt/V	0.28	-0.19 to 0.75	0.24
residual GFR	-0.10	-0.24 to 0.05	0.18
NPNA	0.44	-0.57 to 1.46	0.39
FEBM	-0.01	-0.03 to 0.02	0.69
CF-PWV	-0.06	-0.16 to 0.03	0.20
Overhydration	0.03	-0.03 to 0.10	0.31
Serum CRP	-0.01	-0.01 to 0.004	0.30
Total cholesterol	0.07	-0.10 to 0.24	0.43

Abbreviations: CCI, Charlson's comorbidity index; CF-PWV, carotid-femoral pulse wave velocity; CI, confidence interval; CRP, C-reactive protein; FEBM, fat-free edema-free body mass; GFR, glomerular filtration rate; MTAC, mass transfer area coefficient of creatinine; NPNA, normalized protein nitrogen appearance.

[a]Data of hospitalization were adjusted to the duration of follow-up and then log-transformed for the construction of the linear regression model.[37]

design, we did not study the iron status of our patients, which has been reported to be associated with plasma omentin-1 level in a previous study in hemodialysis patients.[37]

Our present study may provide some insight into the pathophysiology and biology of omentin-1. First, PDE omentin-1 levels were substantially lower than plasma levels and had a modest correlation with peritoneal transport characteristics but not any clinical parameter, suggesting that omentin-1 in PDE originates from the systemic circulation via passive diffusion. Second, plasma omentin-1 level, but not its adipocyte mRNA expression, was associated with clinical outcome, indicating that omentin-1 acts in the traditional endocrine manner on distant targets. Because plasma omentin-1 level only had a marginal correlation with adipose tissue mass, adipose tissues in different body compartments probably do not produce omentin-1 to a similar extent. Furthermore, we also

found that plasma omentin-1 level did not correlate with its adipocyte mRNA level, but the extreme variability of adipocyte omentin-1 mRNA level makes the interpretation of the gene expression result difficult.

There were several notable limitations of our present study. First, omentin-1 levels were measured only at the initiation of PD. With progressive weight gain in PD patients,[8] it would be interesting to explore the corresponding changes in plasma omentin-1 level and its prognostic significance. Unfortunately, because of the limitation in our original study design, we do not have the plasma omentin-1 level during follow-up or the longitudinal trend of many clinical parameters for the exploration of the relationship between baseline omentin-1 level and their subsequent changes. Although omentin-1 is an adipokine linked to systemic inflammation,[13,14] we did not perform an elaborate assessment of systemic inflammation because of the limitations in our original study design. More importantly, our study did not distinguish the causes of hospitalization. Since we found that plasma omentin-1 was associated with the number of hospital admissions, and it has been implicated that omentin-1 is linked to the pathogenesis of the cardiovascular disease,[42,43] it would be interesting to explore whether hospital admission for cardiovascular disease is specifically associated with plasma omentin-1 level.

In addition, there are several areas warranting future research. The underlying mechanisms of cardiovascular protection by omentin-1 in the PD population need to be clarified. The effect of the weight reduction program on the omentin-1 level in obese PD patients and its subsequent clinical impact is another interesting area to explore.

In conclusion, a higher plasma omentin-1 level quartile is not associated with patient or technique survival but is associated with a lower number of subsequent hospital admissions and shorter duration of hospital stay in incident PD patients.

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Do plasma levels of omentin-1 correlate with patient or technique survival among incident peritoneal dialysis patients?

Kidney Medicine

Methods and Cohort

Retrospective analysis of prospective cohort

Incident PD patients N = 152

Duration of follow-up: 5 years

Omentin-1 levels at PD initiation
No repeat measurements

Outcomes and Findings

Plasma Omentin-1 had a modest correlation with:

Hospital admission

and

Duration of stay

p = 0.07
After adjustment for confounders:
p = 0.05

p = 0.04
After adjustment for confounders:
p = 0.03

Plasma Omentin-1 had <u>no</u> correlation with:

Patient survival

and

Technique survival

p = 0.92

p = 0.83

Conclusion: Plasma omentin-1 level was not associated with patient survival, technique survival, or peritonitis, but it was associated with hospital admissions and duration of hospitalization in incident PD patients.

Reference: Than WH, Chan GC-K, Kwan B C-H, et al. Omentin-1 levels and outcomes in incident peritoneal dialysis patients. *Kidney Medicine*, 2023.

Visual Abstract by Jana Sharara

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