WAIST CIRCUMFERENCE AS A PREDICTOR OF ADIPONECTIN LEVELS IN PERITONEAL DIALYSIS PATIENTS: A 12-MONTH FOLLOW-UP STUDY

Ana Paula Bazanelli,¹ Maria Ayako Kamimura,^{1,2} Maria Eugenia Fernandes Canziani,²
Silvia Regina Manfredi,² and Lilian Cuppari^{1,2}

Nutrition Program¹ and Division of Nephrology,² Federal University of São Paulo, São Paulo, Brazil

◆ **Objectives:** This prospective study, conducted at the dialysis unit of the Nephrology Division, Federal University of São Paulo–Oswaldo Ramos Foundation, Brazil, aimed to evaluate whether waist circumference (WC) can predict adiponectin levels in patients undergoing peritoneal dialysis (PD).

◆ **Methods:** Among 115 patients on PD at a single dialysis center who were evaluated at 6 and 12 months, 57% were men, 31% had diabetes, mean age was 52.8 ± 16.1 years, body mass index was 25 ± 4.3 kg/m², and dialysis vintage was 13 months (range: 5 – 33 months). We measured WC at the umbilicus level. Adiponectin was determined by an enzyme-linked immunosorbent assay.

◆ **Results:** At baseline, WC was inversely associated with adiponectin ($r = -0.48$, $p < 0.01$). After adjustment for sex, age, diabetes, peritoneal clearance, and residual renal function, WC was an independent determinant of serum adiponectin ($\beta = -0.52$; 95% confidence interval: -0.73 to -0.31 ; $p < 0.001$). In the prospective analysis, after adjustment for confounders, changes in WC predicted changes in adiponectin. For each unit increase in WC, adiponectin declined by 0.39 mg/L ($p < 0.001$).

◆ **Conclusions:** This study demonstrates that WC is associated with adiponectin and, more importantly, that this simple marker of central adiposity was able to predict changes in adiponectin levels over time.

been described as one of the most important determinants of serum adiponectin in the general population as well as in patients with chronic kidney disease (CKD). In fact, a number of studies have demonstrated the inverse relationship between adiponectin and parameters of body fat (2–8). More specifically, accumulation of fat in the abdominal region appears to significantly affect serum adiponectin levels (6,8). It seems that subcutaneous and visceral fat tissue in the abdomen are both important for lowering adiponectin secretion (9). In a recent study, Zoccali *et al.* (10) demonstrated that abdominal fat, assessed by waist circumference (WC), is a key modifier of the relationship between adiponectin and mortality in a large cohort of hemodialysis patients.

Among the simple methods used to assess abdominal fat, the anthropometric measure of WC, compared with either computed tomography (visceral and subcutaneous abdominal fat) or dual-energy X-ray absorptiometry (trunk fat), has been shown to be a reliable marker of total abdominal fat in various populations (11–14), including CKD patients (15). In patients undergoing peritoneal dialysis (PD), we recently confirmed that WC is a reliable marker of abdominal adiposity (16).

There is evidence showing that abdominal fat accumulates in PD patients over time (17,18), but few studies have investigated the association of central adiposity with circulating adiponectin levels in such patients. We therefore aimed to investigate whether WC is able to predict adiponectin levels in a cohort of PD patients.

METHODS

PARTICIPANTS

Patients were recruited from August 2004 to July 2008. From a total of 126 patients currently treated at our dialysis unit, 115 (41 on continuous ambulatory PD and 74 on automated PD) were eligible and agreed to participate in this study. Only patients 18 years of age or

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Secreted exclusively by adipocytes, adiponectin is a 30-kDa protein with anti-atherogenic, anti-inflammatory, and insulin-sensitizing properties (1). Body fat has

Correspondence to: L. Cuppari, Department of Medicine, Division of Nephrology, University Federal of São Paulo, Rua Pedro de Toledo, 282, São Paulo, SP, CEP 04039-000 Brazil.

lilian@dis.epm.br

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older, who had been on PD for more than 3 months and who had been free of peritonitis for at least 3 months before enrollment with no catabolic conditions, were included in the study. Exclusion criteria included treatment with corticosteroids or immunosuppressive drugs and the presence of ascites or malignant disease.

The study was approved by the university's Ethical Advisory Committee, and informed consent was obtained from each subject.

STUDY DESIGN AND PROTOCOL

In this prospective observational study, all patients initially underwent a first interview to review the inclusion criteria and to obtain informed consent. Subsequently, within 1 – 3 weeks, the patients were admitted to the clinic after 12 hours of fasting and with an empty abdominal cavity to undergo laboratory tests and a nutrition evaluation. All measurements were repeated after 6 months and 12 months. During follow-up, 8 patients died, 8 were switched to hemodialysis, 4 received a kidney graft, 4 withdrew consent, and 11 patients missed the 12-month assessment of WC.

WC AND NUTRITION EVALUATION

We used a flexible plastic tape to measure WC at the patient's umbilicus level at the end of an expiration, while the patient, with empty abdominal cavity, was standing with weight equally distributed on both feet and head facing straight forward. The mean of 3 separate measures was used for analysis. All WC measurements were performed by a single well-trained observer.

Body mass index (BMI) was calculated as body weight divided by height squared (19). Body composition was determined by dual-energy X-ray absorptiometry using a scanner DPX model (Lunar Radiation, Madison, WI, USA), with the patient in the supine position. Additionally, nutrition status was evaluated using the 7-point subjective global assessment (SGA) questionnaire (20). Patients with SGA scores below 6 were considered to be malnourished.

LABORATORY PARAMETERS

Blood samples were drawn after an overnight fast of 12 hours. Serum and dialysate creatinine and urea were determined using a standard autoanalyzer. Serum albumin was measured using the bromocresol green method, and high-sensitivity C-reactive protein (CRP), using immuno-chemiluminescence. Aliquots of serum were frozen and, using commercially available ELISA kits,

adiponectin and leptin (Linco Research, St. Charles, MO, USA) and interleukin-6 (BD Biosciences, San Jose, CA, USA) were determined.

Patients were instructed to collect 24-hour dialysate and urine for measurements of weekly total Kt/V urea, weekly peritoneal creatinine clearance (CCr), and residual renal function. Peritoneal glucose absorption was calculated by subtracting the amount of 24-hour drained glucose from the 24-hour total glucose in instilled dialysate. The transport characteristics of the peritoneal membrane were assessed using a standard peritoneal equilibration test as described by Twardowski (21). Patients were then classified as high, high-average, low-average, or low transporters. High and high-average and low and low-average transporters were combined into "high" and "low" transport groups for analysis.

STATISTICAL ANALYSIS

Data are expressed as mean $\pm$ standard deviation for normally distributed variables and as median and interquartile range for skewed variables. Values for dialysis vintage, CRP, and interleukin 6 were transformed into natural logarithms for analyses. The chi-square test or Student t-test for independent samples was applied as appropriate. The Pearson correlation test was used to evaluate variables associated with serum adiponectin. Determinants of adiponectin at baseline were evaluated using linear regression models that included variables correlating with adiponectin or known to influence adiponectin levels. Mixed models for repeated measures analysis was used to assess changes in laboratory, dialysis, and nutrition parameters over time. Factors associated with changes in adiponectin levels over time were examined by applying a mixed regression analysis. A *p* value less than 0.05 was considered statistically significant. Most analyses were conducted using the SPSS software application (version 15.0: SPSS, Chicago, IL, USA); the mixed regression analysis was performed using the SAS software application (version 9.2: SAS Institute, Cary, NC, USA).

RESULTS

Table 1 depicts the demographic, nutrition, and laboratory characteristics of the 115 study patients. The causes of CKD were diabetic nephropathy (22.6%), hypertensive nephrosclerosis (22.6%), polycystic kidney disease (6.1%), chronic glomerulonephritis (9.6%), undetermined (27.8%), and other (11.3%). In 49% of the patients, BMI was between 18.5 kg/m² and 25 kg/m². Another 47% of the patients were overweight or obese,

and 3% were underweight. On the basis of the values obtained from the SGA, 13 patients (11.3%) were reported to be malnourished (SGA score: 4.5 ± 0.9). Serum adiponectin was significantly lower in patients with diabetes (29.9 ± 12.7 mg/L vs 35.6 ± 13.1 mg/L, $p = 0.03$), in those with a BMI of 25 kg/m^2 or more (28.1 ± 11.7 mg/L vs 39.1 ± 12.1 mg/L, $p < 0.001$), and in those with residual renal function (32.1 ± 13.0 mg/L vs 37.4 ± 12.9 mg/L, $p = 0.04$). Serum adiponectin in the low and high transporter groups (36.2 ± 10.4 mg/L vs 34.3 ± 13.6 mg/L respectively, $p = 0.52$) and in the malnourished and well-nourished patients (35.6 ± 13.0 mg/L vs 33.6 ± 13.2 mg/L respectively, $p = 0.60$) did not differ.

Adiponectin correlated negatively with residual renal function ($r = -0.22$, $p = 0.02$), serum albumin ($r = -0.33$, $p < 0.01$), BMI ($r = -0.47$, $p < 0.01$), body fat ($r = -0.39$, $p < 0.01$), trunk fat ($r = -0.44$, $p < 0.01$), and WC

[$r = -0.48$, $p < 0.01$, Figure 1(A)]. No correlation was found between adiponectin and the other variables studied. Leptin correlated positively with BMI ($r = 0.60$, $p < 0.01$), body fat ($r = 0.76$, $p < 0.01$), trunk fat ($r = 0.70$, $p < 0.01$), and WC ($r = 0.54$, $p < 0.01$). Waist circumference was correlated with body fat ($r = 0.79$, $p < 0.01$) and trunk fat ($r = 0.82$, $p < 0.01$).

As can be seen in Table 2, adjusting for sex, age, diabetes, peritoneal CCr, and residual renal function, WC was independently associated with serum adiponectin levels.

To evaluate longitudinal associations, measurements of adiponectin and WC were also taken at 6 and 12 months. During follow-up, no change in average values was observed for anthropometric, body composition, laboratory, or dialysis parameters (Table 3). However, at the end of the study, a reduction in residual renal function and in peritoneal CCr were observed in 56%

TABLE 1
Demographic, Laboratory, and Nutrition Data of the Patients at Baseline

Variable	Value ^a
Patients (n)	115
Age (years)	52.8 ± 16.1
Dialysis vintage (months)	
Median	13
Range	5–33
Diabetes [n (%)]	36 (31.3)
High transporters [n (%)]	27 (23.5)
Absorbed glucose (g/day)	99.1 ± 43.7
Total Kt/V _{urea}	2.40 ± 0.72
Peritoneal CCr (L/week)	36.2 ± 16.6
RRF (yes) [n (%)]	75 (65.2)
RRF (L/week)	32.6 ± 34.4
Serum creatinine (mg/dL)	8.4 ± 3.4
Serum albumin (g/dL)	3.8 ± 1.0
C-Reactive protein (mg/dL)	1.44 ± 2.68
Interleukin 6 (pg/mL)	13.6 ± 22.0
Adiponectin (mg/L)	33.5 ± 13.5
Leptin (ng/mL)	
Median	37.7
Range	9.6–82.6
Body weight (kg)	66.2 ± 13.9
BMI (kg/m^2)	25.0 ± 4.3
Body fat (kg)	19.4 ± 9.3
Lean body mass (kg)	45.1 ± 9.1
Trunk fat (kg)	10.5 ± 4.8
Waist circumference (cm)	92.1 ± 11.9

CCr = creatinine clearance; RRF = residual renal function; BMI = body mass index.

^a Mean $\pm$ standard deviation unless otherwise indicated.

Figure 1 — Correlation between serum adiponectin and waist circumference in (A) 115 patients at baseline and (B) 80 patients at 12 months.

and 52% of the patients respectively. We also observed large variability in abdominal adiposity and adiponectin changes. In 60% of the patients, WC increased; in 47%, adiponectin declined. As seen in Figure 1(B), adiponectin was negatively associated with WC at 12 months ($r = -0.40$, $p < 0.01$).

Table 4 depicts the linear mixed regression analysis during follow-up. Adjusting for sex, age, diabetes, peritoneal CCr, and residual renal function, changes in WC were independently associated with changes in adiponectin. For each unit increase in WC, a decline of 0.39 mg/L in adiponectin was observed.

DISCUSSION

Our study demonstrates that WC is a determinant of adiponectin in PD patients. Most importantly, this simple

measure of central adiposity was able to predict changes in adiponectin over time.

Kidney seems to be the main organ participating in the biodegradation and elimination of adiponectin from the circulation (22). Accordingly, many investigators have shown that serum adiponectin increases with impairment of renal function (5,23–25). A number of studies have demonstrated that adiponectin is markedly elevated in CKD, particularly in patients on dialysis (26–29). In the present study, and in a previous report in PD patients (29), adiponectin was found to be inversely correlated with renal clearance, confirming that residual renal function can influence serum adiponectin in PD patients.

The dialysis procedure is likely to be inefficient in removing adiponectin, probably because of the high molecular weight of this protein (30 kDa). In fact, a study in PD patients demonstrated that daily removal of

TABLE 2
Multiple Linear Regression Analysis at Baseline Using Serum Adiponectin as the Dependent Variable ($R^2 = 0.34$)

Variable	β	95% Confidence interval	p Value
Sex (female)	5.94	1.15 to 10.7	0.02
Age (years)	0.11	-0.05 to 0.26	0.18
Diabetes (yes)	-0.36	-5.80 to 5.07	0.89
Peritoneal CCr (L/week)	-0.23	-0.43 to -0.03	0.02
RRF (L/week)	-0.10	-1.18 to -0.02	0.01
WC (cm)	-0.52	-0.73 to -0.31	<0.0001
Constant	80.0	49.5 to 110.4	<0.01

CCr = creatinine clearance; RRF = residual renal function; WC = waist circumference.

TABLE 4
Mixed Models Linear Regression Using Serum Adiponectin at Baseline, 6 Months, and 12 Months as the Dependent Variable

Variable	Coefficient	95% Confidence interval	p Value
Sex (male)	-6.45	-10.92 to -1.97	<0.01
Age (years)	0.11	-0.03 to 0.25	0.13
Diabetes	-1.76	-6.60 to 3.09	0.47
Peritoneal CCr (L/week)	-0.11	-0.22 to -0.009	0.03
RRF (none)	3.73	0.09 to 7.37	0.04
WC (cm)	-0.39	-0.56 to -0.23	<0.0001

CCr = creatinine clearance; RRF = residual renal function; WC = waist circumference.

TABLE 3
Anthropometric, Biochemical, and Dialysis Parameters^a of the Patients During Follow-Up

Variable	Baseline	6 Months	12 Months	p Value
Patients (n)	115	90	80	
BMI (kg/m^2)	25.0 $\pm$ 4.3	25.3 $\pm$ 4.4	25.5 $\pm$ 4.1	0.66
Body fat (kg)	19.4 $\pm$ 9.3	20.3 $\pm$ 9.4	20.0 $\pm$ 8.3	0.73
Trunk fat (kg)	10.5 $\pm$ 4.8	11.1 $\pm$ 4.9	11.0 $\pm$ 4.3	0.66
WC (cm)	92.1 $\pm$ 11.9	94.4 $\pm$ 12.3	95.0 $\pm$ 11.8	0.22
Adiponectin (mg/L)	33.8 $\pm$ 13.2	33.8 $\pm$ 13.1	32.8 $\pm$ 13.4	0.84
Peritoneal CCr (L/week)	36.2 $\pm$ 16.6	38.2 $\pm$ 15.8	35.1 $\pm$ 14.3	0.42
RRF (L/week)	32.6 $\pm$ 34.4	27.7 $\pm$ 30.4	23.0 $\pm$ 28.8	0.19

BMI = body mass index; WC = waist circumference; CCr = creatinine clearance; RRF = residual renal function.

^a Expressed as mean $\pm$ standard deviation. A mixed models for repeated measures analysis was used to assess changes in laboratory, dialysis, and nutrition parameters over time.

adiponectin is minimal (29). However, we observed that reduction in peritoneal clearance was also independently associated with accumulation of serum adiponectin, suggesting that, to some extent, adiponectin can be removed by PD.

Corroborating previous studies (4,6,8), we found that central adiposity played an important role in determining serum adiponectin in PD patients. The finding that adiponectin declined in response to abdominal fat gain is of special interest for the PD population, because this group of patients tends to accumulate abdominal fat over time. In patients initiating PD, Fernström *et al.* (17) demonstrated an increase of visceral fat during 15 months of follow-up. A more recent study showed that visceral and subcutaneous fat both accumulated in PD patients during the first 6 months on therapy (18). In the present study, 60% of the patients experienced an increase in abdominal adiposity by WC.

Excess fat in the abdominal region has been noted as a risk factor for metabolic disturbances and cardiovascular events in the PD population (30). Thus, a reliable, easy to perform, and low-cost method for assessing abdominal fat is relevant to the clinical management of PD patients. Although WC has been well established and widely used as a measure of central adiposity in various populations (11–14), only recently has it been tested in CKD patients (15). In a cohort of 122 non-dialyzed CKD patients, WC was strongly associated with abdominal visceral and subcutaneous fat as determined by computed tomography (15). More importantly, WC was associated with cardiovascular risk factors in the same manner that visceral and subcutaneous fat by computed tomography was (15). In a very recent prospective study, we demonstrated that WC is a reliable marker of abdominal adiposity in PD patients (16). Thus, this simple anthropometric measure seems to be useful in the routine care of PD patients, even considering the particularities of the PD modality (such as abdominal distension) that might compromise measurement of the abdominal perimeter.

As mentioned, abnormal fat deposition in the abdominal region determined by sophisticated methods is linked to metabolic abnormalities in PD patients (30). However, few data are available on the relationship between WC and markers of metabolic disturbance, adipokines in particular, in the dialysis population (10,31). Adiponectin, leptin, interleukin 6, and tumor necrosis factor α are proteins synthesized in adipose tissue that have important endocrine functions in CKD and may also be important contributors to systemic inflammation in that population (32). Among them, adiponectin—a protein with several beneficial cardiovascular properties—has gathered interest because its synthesis is blunted with

the abnormal accumulation of adipose tissue mainly in the abdominal region (6,8). Accordingly, in the present study, adiponectin correlated more strongly with trunk fat and WC than with total body fat.

Although a study has already shown the inverse association between WC and adiponectin in patients on PD (31), our investigation extends that work by demonstrating for the first time that changes in WC over time can predict changes in adiponectin. In combination with the present results, our previous finding that WC is a reliable marker of abdominal adiposity strengthens the usefulness of this simple method in the care of PD patients.

Given that adiponectin metabolism is dysregulated in CKD patients because of reduced clearance and probably because of the effects of persistent inflammation, the impact of adiponectin on outcomes in this population is still a matter of debate. Several earlier reports demonstrated that low adiponectin is related to cardiovascular events and mortality (27,33–35), but recent studies showed that high, and not low, adiponectin predicts poor outcome (36,37). These controversial findings may be explained, at least in part, by differences in the degree of adiposity of the study patients. Indeed, in a recent study with a large cohort of hemodialysis patients, the predictive value of adiponectin for all-cause and cardiovascular death was shown to depend on WC (10). The authors demonstrated that the 4-year probability of death for increased adiponectin was high among patients with a small WC and low in patients with a large WC. In PD patients, studies to confirm whether WC should be taken into account as a valid interpretation of the association between adiponectin and mortality are required.

CONCLUSIONS

In the present study, we demonstrated that WC is associated with adiponectin and, most importantly, that this simple marker of central adiposity is able to predict changes in adiponectin over time. Although our study is limited by a relatively small sample size, the prospective design strengthens the association between this simple anthropometric marker of abdominal adiposity and an important circulating adipokine. The predictive value of WC on outcomes and the role of WC in modifying the association between risk factors and morbidity and mortality remain to be investigated in the PD population.

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DISCLOSURES

All authors have no financial conflicts of interest to declare.

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