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Adiponectin in Children on Peritoneal Dialysis: Relationship to Insulin Resistance and Nutritional Status

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Key Words
Adiponectin, children · Chronic kidney disease · Continuous ambulatory peritoneal dialysis · Insulin resistance · Nutritional status · Oral glucose tolerance testing · Peritoneal dialysis

Abstract
Aim: To study whether adiponectin and resistin serum concentrations in children on peritoneal dialysis (PD) were related to insulin resistance (IR) and anthropometric parameters of nutritional status, 11 PD patients, 9 chronic kidney disease (CKD) patients and 10 healthy children were studied. ***Methods:*** Glucose and insulin were measured during the oral glucose tolerance test. Levels of adiponectin and resistin were evaluated by ELISA, insulin by RIA. ***Results:*** In CKD patients, higher homeostasis model assessment-insulin resistance (HOMA-IR), fasting and 2-hour serum insulin levels were shown compared to control and to PD patients. Body mass index (BMI) and body fat content were severely decreased while serum adiponectin levels were significantly higher in PD patients relative to controls. No differences among groups were shown in resistin levels. On regression modeling, inverse independent associations were observed between adiponectin with percentile BMI, weight and height z-score, and with body fat content. In contrast, no relationship was found between adiponectin and IR parameters. In multiple regression analysis, adiponectin was negatively correlated with BMI. A negative association of adiponectin and resistin with glomerular filtration rate was also shown. ***Conclusion:*** A role for adiponectin in terms of its association with clinical wasting parameters in PD pediatric patients might be suggested.

Introduction

Patients with chronic kidney disease (CKD) have metabolic, nutritional and inflammatory alterations which in synergic combination act as a source of oxidative stress and vascular injury. Insulin resistance (IR) in renal patients is accompanied by hyperinsulinemia and glucose intolerance as well as by complex derangements of insulin secretion [1–3]. Increasing evidence has associated IR with subclinical inflammation involving cytokines derived from adipose tissue [4]. Adipose tissue is a highly active endocrine organ that secretes a wide range of bioactive peptides, known as adipokines with autocrine, paracrine and endocrine actions. An adipose tissue-derived protein, adiponectin, is closely related to glucose and lipid metabolism. It is known to reduce IR in humans. A potential insulin-sensitizing function of angiotensin II type 1 receptor blockade with regard to increased

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adiponectin has been described, although so far it is not fully explained [5, 6].

Induced during adipocyte differentiation, adiponectin secretion is stimulated by insulin [7–9]. Additionally, adiponectin regulates energy homeostasis. Recently, this adipokine has been suggested to prevent atherosclerosis, being an effective anti-inflammatory protein for vascular walls [10, 11]. On the other hand, resistin, a cystine-rich 108-amino-acid peptide hormone, is a newly discovered peptide hormone that inhibits adipogenesis [12, 13]. Resistin antagonizes insulin action and has been shown to impair glucose metabolism [14]. It may be involved in regulative processes taking place in IR and inflammation [15]. However, in humans there is uncertainty about the relationship among resistin, body fat mass and IR. Despite numerous clinical studies regarding insulin sensitivity of CKD patients, little data is available concerning the effect on continuous ambulatory peritoneal dialysis (CAPD) therapy in pediatric patients.

A significant number of patients with peritoneal dialysis (PD) develop an increased permeability for small solutes, which induces a faster absorption of glucose causing a chronic stimulation of insulin secretion [16]. Protein energy malnutrition can often lead to growth delay and extremes in body mass index (BMI) which in turn have been associated with increased morbidity and mortality in pediatric patients on PD [17, 18]. Characterized by a synergistic combination of anorexia, increased basal metabolic rate, loss of lean body mass and declining serum proteins, these groups of nutritional abnormalities described as disease-associated wasting are present in pediatric PD patients [19]. Although the mechanisms underlying disease-associated wasting are not well understood, adipokines may play an important role given that they mediate IR and inflammation in kidney disease [20].

The aim of this study was to examine whether the changes in adiponectin and resistin serum levels may be related to IR and nutritional status evaluated by anthropometric parameters, in pediatric patients on PD treatment.

Material and Methods

Patients

Twenty patients, 8 boys and 12 girls, aged 11.4 ± 3 (range 5–14.5) years, were studied. These included 9 stage 3–4 CKD patients aged 12.2 ± 2.6 years (group I) and 11 PD patients aged 10.4 ± 3 years (group II). Twelve healthy children, matched for age, sex and BMI patients' brothers and sisters, aged 9.2 ± 3.9, were recruited as controls (group III).

This study protocol was duly approved by the Ethical Committee of 'Dr. Humberto Notti' Pediatric Hospital, and informed consent was obtained from all children's parents after explaining the purpose of the study. The etiology of CKD and PD patients included chronic glomerulonephritis in 4 patients, hemolytic uremic syndrome in 3 patients, interstitial nephritis in 1 patient and renal dysplasia/obstructive uropathy in 12 patients.

None of the patients were currently on or had been on corticosteroids. Obese patients (BMI >25 kg/m^2) in the control group were not included. Malignancy, infection, or obvious inflammation was not diagnosed in the two groups of subjects, and all subjects continued their regular medication. For the last 12 months, 10 PD patients had no episode of peritonitis, only 1 peritonitis episode was registered in a patient, 5 months before the study.

The PD patients used either CAPD with the twin-bag system (Ultrabag; Baxter, McGraw Park, Ill., USA) or continuous cyclic PD with a cycler (HomeChoice; Baxter), with a mean treatment duration of 3.5 ± 1.02 years.

Clinical Measurements

Anthropometric data were obtained once, for each subject at enrolment in the study and involved evaluation of height, weight and BMI. BMI (kg/m^2) was calculated from body weight and height. The height-specific BMI reference values adjusted to age was shown. The variation in BMI due to variation in height at each age was evaluated through the 3rd, 5th, 50th, 85th, 95th and 97th percentiles [21]. In addition, in the three groups, weight-to-height indexes were performed.

In order to express the deviation in standard deviation (SD) units from the Argentine reference curves for age and gender [22], z-scores were used. Individual weight and height SD scores (z) were calculated according to the following equation: z score = x – mean value for normal/SD for normal children, where x is the value of an individual patient and SD is the standard deviation for the normal population at the same chronological age.

Triceps and subscapular skinfold thickness measurements were performed in triplicate by using a Holtain caliper (Holtain Ltd., Crosswell, Crymych, UK). The results were plotted against chronological age and expressed in percentiles [23]. Blood pressure was taken according to Task Force recommendations [24]. Nine patients from groups I and II were treated with antihypertensive drugs at the time of the study: angiotensin-converting enzyme inhibitor (IECA) and/or amlodipine.

Analytical Procedures

Oral Glucose Tolerance Testing

Children underwent the oral glucose tolerance test (OGTT), which is the most commonly used method to evaluate whole-body glucose tolerance in vivo [25]. After an 8- to 10-hour overnight fast, blood samples were drawn for the determination of baseline glucose and insulin. After the children from the three groups orally received glucose (1.75 g/kg), blood samples were taken at 30, 60 and 120 min for the measurement of plasma glucose and insulin concentrations. No dialysis was performed in the PD group of patients the night before the test. Area under curve (AUC) for glucose and insulin serum concentration after glucose charge, basal homeostasis model assessment of insulin resistance (HOMA-IR) and insulinogenic index were used as markers of tissue insulin sensitivity. Fasting morning blood samples were also taken in the three groups of patients for measurement of lipid pro-

Table 1. Demographic, anthropometric, and biochemical data for patients with CKDs, patients undergoing PD, and healthy children

	CKD patients	PD patients	Healthy children
Male/female	3/6	5/6	3/7
Age, years	12.2 ± 2.6	10.4 ± 3	9.2 ± 3.9
BMI percentile	46.11 ± 8.74	23.36 ± 10.24 [a]	63.00 ± 7.64
Weight to height ratio, %	95.85 ± 3.17	91.53 ± 3.59[b]	97.62 ± 2.76
Height z-score	–1.57 ± 0.44 (–4.01 to 0.10)[b]	–1.99 ± 0.35 (–3.81 to –0.45)[c]	0.26 ± 0.25 (0.02–1.41)
Weight z-score	–1.28 ± 0.4 (–3.68 to 0.14)[a]	–1.61 ± 0.3 (–3.57 to –0.4)[b]	0.82 ± 0.4 (–1.33 to 3.87)
GFR, ml/min/1.73 m^2	29.82 ± 5.9[c]	8.95 ± 0.73[c]	116.1 ± 2.63
Triglycerides, mg/dl	194.71 ± 21.26[b]	181.90 ± 26.12[b]	75.12 ± 5.45
HDL cholesterol, mg/dl	50.50 ± 4.73	55 ± 2.75	55.12 ± 3.69
LDL cholesterol, mg/dl	95.51 ± 2.84	130.30 ± 16.01[b]	72.37 ± 4.34
Cholesterol, mg/dl	171.55 ± 2.87	211.54 ± 16.32[b]	134.55 ± 6.20
HOMA-IR	2.28 ± 0.26[c]	1.31 ± 0.16[d]	0.95 ± 0.18
Insulinogenic index	0.90 ± 0.12	3.63 ± 2.33	5.38 ± 4.46
Adiponectin, μg/dl	25.50 ± 1.99[d]	39.38 ± 3.38	26.36 ± 3.36[b]
Resistin, ng/ml	17.43 ± 1.20	18.29 ± 1.20	13.93 ± 1.19

Values expressed as mean ± SEM. Statistical significance is from one-way ANOVA.
[a] $p < 0.05$ PD patients vs. control group. [b] $p < 0.01$ PD patients vs. control group. [c] $p < 0.001$ PD patients vs. control group. [d] $p < 0.01$ PD patients vs. CKD group.

files, creatinine determination, and for the determination of serum levels of the adipokines, adiponectin and resistin.

Assay and Calculations

Glomerular filtration rate (GFR) was calculated by the Schwartz formula [26]. All PD children had a creatinine clearance <10 ml/min which was adequate with PET and urea Kt/V following DOQI guidelines. Serum glucose levels were determined by the glucose-oxidase-peroxidase technique [27]. Enzymatic in vitro assay for the direct quantitative determination of HDL cholesterol and LDL cholesterol in human serum were performed on a Hitachi 912 automated chemistry analyzer (Roche, Mannheim, Germany). Serum insulin concentrations were measured by RIA by using reagents from Diagnostic Products Corp. (Los Angeles, Calif., USA). HOMA-IR was calculated using the following formula: glucose (mM) × insulin (mIU/l)/22.5. Insulinogenic index was calculated: Δ insulin (30 min – 0 min)/Δ glucose (30 min – 0 min). The AUC for plasma glucose and plasma insulin obtained every 30 min for a 2-hour period was calculated per subject using the trapezoidal rule.

Adiponectin and Resistin Concentrations

Adiponectin and resistin were measured using an enzyme-linked immunosorbent assay kit (ELISA) (Chemicon International, Temecula, Calif., USA). Bound adiponectin and resistin were captured by biotinylated anti-human adiponectin and resistin polyclonal antibodies. Horseradish peroxidase-conjugated streptavidin was added. The sensitivity for adiponectin and resistin was 100 pg/ml. The intraassay variation was 3.84% and interassay variation was 5.5% for adiponectin, and the intra- and interassay coefficients of variation for resistin were 5.55 and 7.20%, respectively.

Statistical Analyses

The results were assessed by one-way analysis of variance to compare the three groups. Significance of differences between groups was estimated by the Bonferroni post-test. The associations between variables were assessed by Pearson correlations. Stepwise forward multiple regression analysis was performed to assess independent predictors of serum adiponectin. A p value <0.05 was considered statistically significant.

Results

Patients' Characteristics

The anthropometric characteristics of the three groups of children are summarized in table 1. Decreased percentile values of BMI in the PD group of patients versus control children are shown ($p < 0.05$) (table 1). Of the 11 PD patients we studied, percentile values of BMI were below the 50th percentile in 8 children and in 6 children below the 15th percentile. In the CKD group, height-specific BMI below the 50th percentile was demonstrated in 2 patients. A significantly lower weight/height relation was shown in PD patients when it was compared to CKD patients and controls (both $p < 0.01$) (table 1). Moreover, a decreased mean height z-score and weight z-score were demonstrated in PD patients compared to healthy children ($p < 0.001$ and $p < 0.01$, respectively). Lower values in mean height z-score were also shown in CKD patients

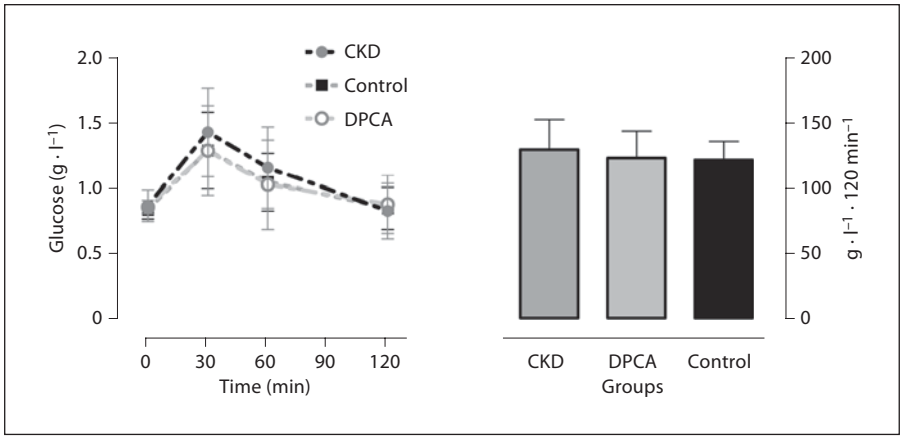

Fig. 1. OGTT and AUC for plasma glucose in patients undergoing PD, patients with CKDs and control children. Right: Serum glucose concentrations during an OGTT performed in PD patients in CKD patients and control children. Left: AUC for glucose is shown.

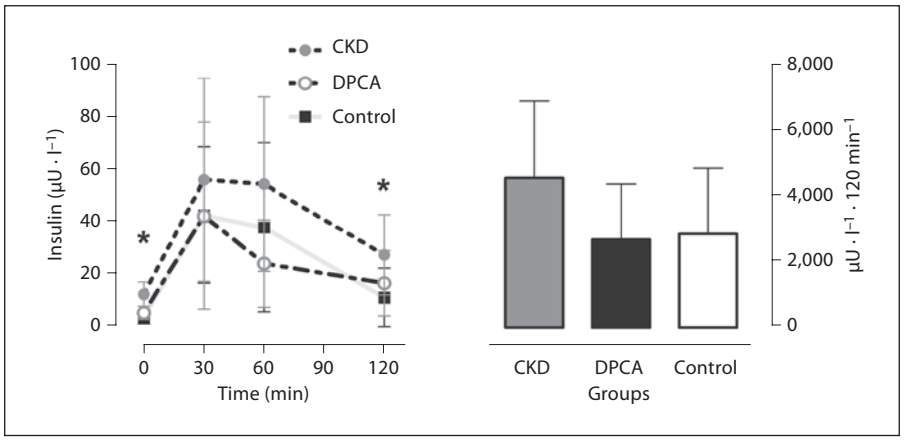

Fig. 2. OGTT and AUC for plasma insulin in patients undergoing PD, CKD patients and control children. Right: Serum insulin concentrations during an OGTT performed in PD patients, in CKD patients and control children. CKD patients had significantly higher fasting and 2-hour insulin levels compared to control (* $p < 0.01$) and to PD patients (* $p < 0.05$). Left: AUC for insulin is shown.

compared to controls ($p < 0.01$) (table 1). Children on PD treatment showed decreased triceps and subscapular skinfold thickness expressed as percentiles, related to controls: 32.6 ± 9.3 versus 66.5 ± 4.7 ($p < 0.05$) and 33.9 ± 9.2 versus 64 ± 4.8 ($p < 0.05$), respectively.

GFR (ml/min/1.73 m^2) following the Schwartz formula in CKD patients was 29.82 ± 5.9 (range 12–55.7 and 8.95 ± 0.73 ml/min/1.73 m^2 in PD children, respectively). Children on PD treatment had urea Kt/V 3 ± 0.47, creatinine clearance test 152.3 ± 28 l/week/1.73 m^2, and nPCR 2.9 ± 0.21 g/day.

OGTT and AUC for Plasma Glucose and Insulin Results

β-Cell function and insulin sensitivity were studied using HOMA-IR, AUC for glucose and insulin, and also by the insulinogenic index. The serum glucose levels during the OGTT in CKD, PD and control children are presented in figure 1. Fasting serum glucose was within normal limits in all patients, moreover no significant difference at 30, 60 and 120 min was demonstrated between groups.

CKD patients had significantly higher fasting and 2-hour serum insulin levels compared to control (both $p < 0.01$). Moreover, higher insulin levels were also demonstrated in CKD patients related to PD patients for fasting insulin ($p < 0.01$) and for 2-hour plasma insulin ($p < 0.02$). The mean AUC serum insulin level was higher in CKD patients although it did not reach a significant difference compared to PD patients and controls (fig. 2).

The HOMA-IR level of group I was significantly higher compared to that of group II ($p < 0.001$) and group III ($p < 0.001$). However, no differences were seen on the insulinogenic index among groups (table 1). Concerning the lipid profile, we found abnormal LDL cholesterol, total cholesterol and triglyceride levels in patients with PD related to the control group (table 1). Serum adiponectin levels were significantly higher in PD patients than in CKD patients and control children (both $p < 0.01$, respectively). In contrast to marked differences in serum adipo-

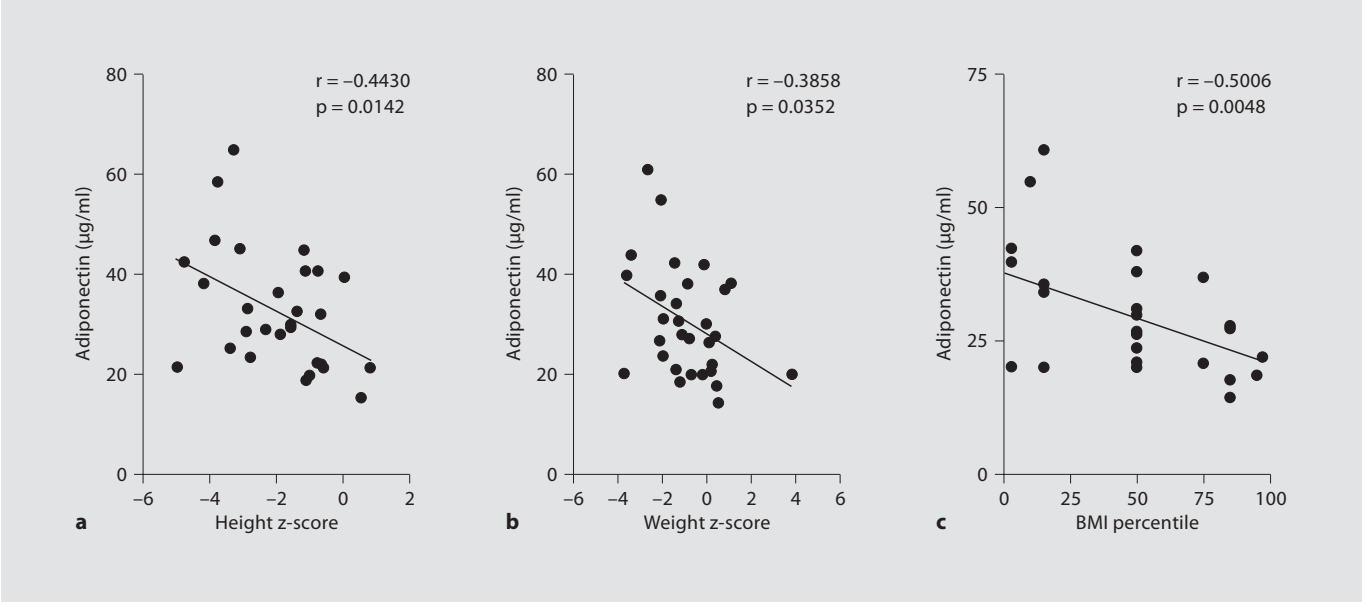

Fig. 3. Relationship between adiponectin levels and percentile values of BMI and weight and height z-scores. An inverse association was observed on bivariate analysis between adiponectin levels and percentile BMI for the groups as a whole (r = –0.50, p = 0.004). An inverse association was observed on bivariate analysis between fasting plasma adiponectin and height and weight z-scores for the groups as a whole (n = 30) (r = –0.44, p = 0.01 and r = –0.38, p = 0.03, respectively).

nectin levels between PD patients with CKD and control children, serum resistin concentrations in PD patients did not differ from those of CKD and control children (table 1).

Relationship between Plasma Adiponectin and Biochemical Parameters

As seen in figure 3, there was also a negative relationship between serum adiponectin with percentile values of BMI (r = –0.50, p = 0.004), height z-score values (r = –0.44, p = 0.01) and with weight z-score values (r = –0.38, p = 0.03) in the three groups of children. Adiponectin was also negatively associated with triceps skinfold thickness (r = –0.61, p = 0.003) and with subcapsular skinfold thickness (r = –0.60, p = 0.004) in the three groups of children. Serum adiponectin was inversely correlated with GFR (r = –0.41, p = 0.02) (fig. 4).

Serum adiponectin levels were related neither to serum HDL cholesterol levels nor to serum triglyceride levels. Also from the analysis using Pearson's correlation, there was no significant relationship between serum adiponectin with HOMA-IR and with resistin levels in the total sample (table 2). Adiponectin levels failed to show a significant association with AUC insulin in CKD patients

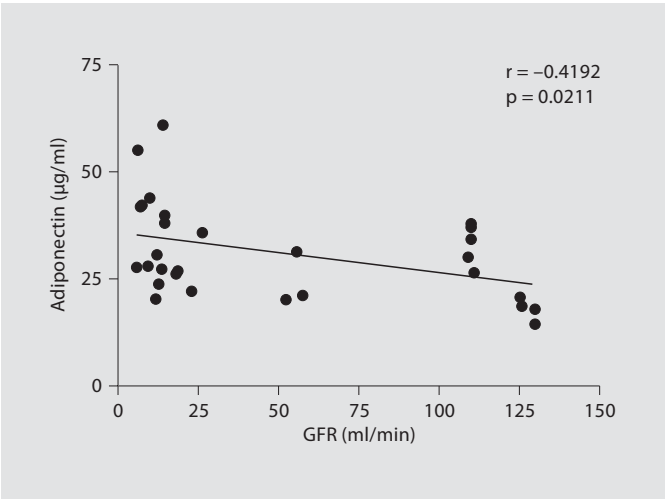

Fig. 4. Relationship between adiponectin levels and GFR. An inverse association was observed between GFR as calculated by the Schwartz formula and fasting serum adiponectin on bivariate analysis (n = 30) (r = –0.42, p = 0.01).

Table 2. Relationship of adiponectin and resistin levels with anthropometric, biochemical and hormonal parameters in patients undergoing PD, patients with CKDs and control children

	Adiponectin	Height z-score	Weight z-score	Percentile BMI	GFR	HDL chol	Triglycerides	HOMA-IR	Resistin
Adiponectin	1.00	r = –0.4430 p = 0.014	r = –0.3852 p = 0.035	r = –0.5006 p = 0.004	r = –0.4192 p = 0.0211	r = 0.0592 p = 0.756	r = 0.2594 p = 0.166	r = –0.1173 p = 0.537	r = 0.1788 p = 0.344
Resistin	r = 0.1788 p = 0.344	r = –0.3328 p = 0.072	r = –0.2320 p = 0.2173	r = 0.0741 p = 0.6969	r = –0.4804 p = 0.007	r = 0.2731 p = 0.144	r = 0.1561 p = 0.411	r = 0.0389 p = 0.838	1.000

Statistical significance is from Pearson's correlation. GFR = Glomerular filtration rate; HDL chol = high-density lipoprotein cholesterol.

Table 3. Multiple linear regression model analysis considering adiponectin as dependent variable

Variable	Coefficient	Standard error	95% confidence interval	t ratio	p value
Constant	40.309	2.874	34.411 to 46.207	14.024	<0.0001
GFR	–0.04096	0.03844	–0.1198 to 0.03791	1.066	0.2960
BMI percentile	–0.1682	0.05783	–0.2868 to –0.04950	2.908	0.0072

(r = –0.39, p = 0.29, n = 9), in PD patients (r = –0.48, p = 0.13, n = 11), and in controls (r = –0.07, p = 0.83, n = 10).

Table 3 shows the results of the stepwise multiple linear regression analysis. There was no multicollinearity between independent variables. Multiple regression analysis indicated that the BMI percentile was inverse related with serum adiponectin levels (p = 0.01), as we have demonstrated by Pearson's correlation analysis. In contrast, GFR did not reach statistical significance (probably because of the sample size), but shows a consistent trend to fit the models. For our results, we found that changes in adiponectin concentration were not a predictive factor for changes in the IR parameters in the studied patients. Correlations between resistin serum levels were unrelated to either anthropometric or insulin sensitivity variables, although the resistin serum concentration was inversely related to GFR (r = –0.4804, p = 0.007) (table 2).

Discussion

The present study shows that adiponectin levels in pediatric patients on PD treatment were strongly related to nutritional status measured by anthropometric parameters, whereas no relationship to insulin resistance was shown. Resistin serum levels were unrelated to either anthropometric components of nutritional status or insulin sensitivity variables.

Despite numerous clinical studies regarding insulin sensitivity of CKD patients, there are little data available concerning the effect of CAPD therapy in pediatric patients. Most of the data on insulin metabolism, which comes from small cross-sectional studies of children on chronic dialysis, have shown that hyperinsulinemia and IR are frequent in children with mild to moderate CKD [28–30]. Recently, the prevalence of hyperinsulinemia in lean pediatric patients with mild to moderate renal dysfunction suggests that there is a deregulation of glucose metabolism, independent of elevated BMI [31].

Although the possibility that glucose loading may worsen insulin sensitivity, our present study showed an absence of IR in PD-treated pediatric patients. No differences were demonstrated after OGTT in AUC for insulin and glucose, nor in HOMA-IR analysis, when PD pediatric patients were compared to controls. In contrast, this study added to previous findings demonstrating that CKD from pediatric patients is accompanied by IR. Fasting insulin and HOMA-IR index were demonstrated to be increased in this group of patients. Although the mean AUC plasma insulin was elevated in CKD patients, it did not reach significant differences.

Favorable results regarding the effect of PD on normalizing IR in uremia have been previously suggested. Moreover, cycling peritoneal dialysis (CCPD) compared to HD has shown that the final insulin sensitivity was higher in the CCPD group [32]. On the contrary, it has

also been reported that patients with CAPD displayed IR [33].

Associations have been shown between adiponectin levels and the components of the insulin resistance syndrome and glucose resistance [34]. Here we showed that PD patients had greater serum adiponectin levels in comparison to CKD patients and controls. An interesting previous finding was that PD patients who absorbed more glucose had greater adiponectin levels. As an insulin sensitizer, adiponectin facilitation of glucose metabolism may increase glucose uptake from dialysate [35]. Although these previous findings suggest that adiponectin actively influences glucose and insulin metabolism, in this study we were unable to identify a relationship between serum adiponectin with AUC glucose, AUC insulin or HOMA-IR.

On the other hand, a significant inverse correlation was found between adiponectin plasma levels and anthropometric components of nutritional status. Adiponectin was independently associated with percentile values of BMI, weight and height z-scores, and also with triceps and subscapular skinfold thicknesses for the three groups as a whole. Moreover, from our results of multiple regression analysis, we found that changes in adiponectin concentration were a predictive factor for changes in percentile values of BMI in the studied patients.

The inverse association between adiponectin and BMI and also with body fat content observed in the pediatric patients of our study is consistent with recent reports in patients after substantial weight loss [36, 37]. A gradual rise in adiponectin levels related to the nutritional status of anorexia nervosa has also been reported [38]. A potentially deleterious effect of high adiponectin levels on nutritional status in advanced CKD adult patients has been previously suggested. This may be due to the possible role of adiponectin in increasing energy expenditure, which in catabolic states such as CKD may lead to accelerated muscle wasting and adverse disease outcomes [39]. In contrast, a protective role of high levels of adiponectin has been suggested in patients with intense weight loss. Hyperadiponectinemia could contribute to the increased insulin sensitivity, because it can inhibit hepatocyte glucose uptake and enhance insulin-stimulated glucose uptake and lipid oxidation in muscle [40]. Moreover, because adiponectin blocks B-lymphopoiesis, suppresses macrophage function and interferes with TNF-α signaling, its elevated levels could contribute to the immune suppression observed in malnourished patients [41]. Furthermore, it was recently suggested that adiponectin might have a role in maintaining energy homeostasis under energy shortage conditions [42]. Notwithstanding, the possible advantages of long-term accumulation of this protein in PD patients is not yet known.

The metabolic pathway for adiponectin is not clear, as renal clearance may have a role. Our study showed that adiponectin levels were significantly related to residual renal function. This observation was compatible with the negative significant correlation we have demonstrated between GFR and adiponectin levels. In a previous study, it was reported that adiponectin, because of its high molecular weight, was not readily removed by dialysis. Renal transplants may decrease adiponectin levels, indicating the kidney's role in the biodegradation of adiponectin [43]. In addition, the lower total adiponectin levels and messenger RNA for adiponectin receptor AdipoR1 after transplants may be secondary to an improvement in GFR [44]. However, our results of multiple linear regression model analysis considering adiponectin as a dependent variable showed that GFR did not reach statistical significance. In contrast, we found a strong negative relationship between adiponectin levels and percentile values of BMI.

Other energy-modulating hormones such as the adipokine resistin have been described to be perturbed in uremia and could contribute to nutritional abnormalities. Circulating resistin did not significantly differ in the groups studied herein and a link between resistin levels with IR or anthropometric variables was not shown. This finding may be in part explained by the fact that although leptin and adiponectin are produced almost exclusively by adipocytes [45], the main source of resistin in humans is immunocompetent cells in the adipose tissue [46].

Experimental data have demonstrated that fat resistin mRNA expression is severely reduced in morbidly obese ob/ob mice, however circulating resistin levels are increased compared to lean animals and thus could contribute to IR phenotype [47]. In contrast, resistin's role in human physiology is currently unclear. There are generally contradictory data regarding resistin levels, ranging from reports showing a positive correlation with BMI to no relationship at all [48, 49]. Kielstein et al. [50] showed that plasma resistin levels increased with the progressive impairment of renal function. As has been previously reported [51], our results demonstrated that with decreasing renal function an increased resistin concentration in serum was shown.

A limitation of this study is the small number of pediatric patients (sample size) and thus the possibility of statistical bias exists. Despite this limitation, we have shown that circulating adiponectin and resistin levels are inverse related to GFR.

Conclusion

This study provides insight into the association of adiponectin levels with nutritional status, showing that in pediatric patients undergoing PD, adiponectin increases as a dependent variable, while anthropometric components of nutritional status decrease. In contrast, adiponectin levels were unrelated to IR. A role of adiponectin in terms of its contribution to malnutrition/disease-associated wasting in PD pediatric patients might be suggested.

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