Early Changes in Bioelectrical Estimates of Body Composition in Chronic Kidney Disease

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The aim of this study was to detect the potential occurrence of early abnormalities of body composition in patients with chronic kidney disease (CKD) at first referral to an outpatient nephrology clinic. Eighty-four patients with CKD (49 men and 35 women) were compared with 604 healthy control subjects (298 men and 306 women). Anthropometry and bioelectrical impedance analysis (BIA) were performed in all participants, whereas renal function, laboratory tests for nutritional status, and nutrient intake were assessed in the CKD group only. Creatinine clearance was 27.8 ± 13.8 and 27.4 ± 13.0 ml/min per 1.73 m^2 in male and female patients with CKD, respectively. No patient showed peripheral edema; frank malnutrition, defined by presence of serum albumin <3.5 g/dl plus body mass index <20 kg/m^2; or protein intake <0.6 g/kg per d. At the BIA, patients with CKD showed lower resistance (R) and abnormal mean impedance vectors for the bivariate normal distribution of R/height and reactance/height. Phase angle also was reduced (−22%), especially in patients with diabetes. When BIA-derived data were considered, total body water was slightly higher (+4.3% in men; +3.5% in women) and body cell mass was lower (−6.7% in men; −7.7% in women) in patients with CKD. No difference in either BIA parameters or nutritional indexes was observed among various CKD stages. Despite the absence of overt malnutrition, patients with CKD exhibit altered BIA variables from the early phases of renal disease. These alterations are related to the renal dysfunction, are more marked in the presence of diabetes, and mainly indicate the presence of overhydration in the absence of edema. Therefore, BIA represents an attractive clinical tool to detect impairment of body composition from the early stages of CKD.

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Malnutrition frequently occurs in patients with ESRD and is associated with increased morbidity and mortality during maintenance dialysis (1–3). Whereas a large body of experimental evidence on nutritional status has been collected in dialysis patients, only a limited number of studies have been performed in the earlier phases of chronic kidney disease (CKD) (4–13).

The progressive reduction of protein and energy intakes (4–6) and other specific factors, such as metabolic acidosis (14), may contribute significantly to the impairment of nutritional status throughout the course of CKD. Abnormalities of nutritional variables, such as serum albumin, body mass index (BMI), and protein intake, that are commonly considered as markers of protein-energy malnutrition (15) usually have been described only in the advanced CKD (4,6,8,9,11,12). In contrast, few data have been provided as to when malnutrition occurs during the course of kidney disease; similarly, whether early to moderate CKD is associated with some abnormalities in nutritional status or body composition remains ill-defined. Such information is crucial to prevent the onset of overt malnutrition and, consequently, to ameliorate the outcome of these patients.

Assessment of body composition is commonly included in the general evaluation of nutritional status. Single-frequency bioimpedance analysis (BIA) is a simple bedside technique that is used for various purposes in patients with renal diseases (16,17). Derived values for fat-free mass (FFM) and total body water (TBW) can be estimated using predictive formulas that include measured BIA variables (16). Alternatively, resistance and reactance *per se* have been used to determine a patient's hydration status (BIA vector analysis) (18). Furthermore, the reduction of phase angle (PhA) has been considered to reflect an increase in the ratio between extra- and intracellular water or a decrease in body cell mass (BCM) (17). From the clinical point of view, it also is noteworthy that PhA is an independent predictor of survival under various pathologic conditions and, in particular, in hemodialysis patients (19,20). At variance with the dialytic phase, in predialysis patients with CKD, the few available papers on BIA have yielded inconsistent results as regards the occurrence of impairment in body composition (11–13,21,22). The aim of this study was to identify early abnormalities in body composition in CKD by means of BIA analysis and assessment of main nutritional markers in predi-

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alysis patients who were at various CKD stages at the first referral to an outpatient nephrology clinic.

Materials and Methods

Participants

Consecutive adult patients who had CKD and were referred for the first time to our outpatient renal clinic were considered for inclusion in the study. Patients were considered eligible when their creatinine clearance (CrCl) was ≤60 ml/min per 1.73 m^2 on two determinations performed 2 wk apart (CKD group) and were divided according to Kidney Disease Outcomes Quality Initiative (K/DOQI) criteria: CKD stage 3, CrCl 60 to 30 ml/min per 1.73 m^2; CKD stage 4, CrCl 30 to 15 ml/min per 1.73 m^2; and CKD stage 5, CrCl <15 ml/min per 1.73 m^2. Exclusion criteria were dialysis treatment; kidney transplant; diagnosis or suspicion of reversible cause of renal failure; clinical conditions that affect body composition, such as cancer, severe liver failure, and alcoholism; habitual use of drugs that influence body composition or renal function (*e.g.*, immunosuppressive or anti-inflammatory drugs); physical amputation; severe obesity (BMI ≥35 kg/m^2); clinically detectable edema; and catabolic conditions. None of the patients selected had acute obstructive causes of renal failure at ultrasound examination. All participants gave their informed consent to the study, and the study protocol was approved by the local Ethics Committee.

The control group consisted of healthy individuals who were matched for gender, age, and BMI and selected among a group of 1632 healthy individuals who were enrolled in a previous general population-based study from our group (23). Individuals who were undergoing heavy physical training (*i.e.*, >1 h of vigorous exercise per day) were excluded.

Measurements and Calculation

Patients with CKD underwent blood sampling early in the morning, after fasting for 10 to 12 h. In the same group, a 24-h urine collection also was obtained to assess renal function and protein intake. Urine collection was considered inaccurate and discarded when creatinine excretion fell outside the 60 to 140% range of the value estimated according to Dwyer and Kenler (24).

Samples were analyzed by standard laboratory methods with serum albumin determined by BromoCresol Purple (normal range 3.5 to 4.8 g/dl). CrCl was normalized to a body surface area of 1.73 m^2. Dietary protein intake was estimated from 24-h urea nitrogen excretion (25).

Anthropometry and BIA

Measurements were performed early in the morning. Weight was measured to the nearest 0.1 kg and height to the nearest 0.5 cm. BMI was subsequently calculated as the ratio weight/height^2 (kg/m^2).

Single-frequency BIA was determined on the nondominant side of the body, in the post absorptive state, injecting 800 μA and 50 kHz alternating sinusoidal current with a standard tetrapolar technique (BIA 101 Impedance Analyzer; Akern, Firenze, Italy). BIA was performed in standardized conditions: A quiet environment, ambient temperature of 22 to 24°C, after voiding, and after being 20 min at rest in the supine position (26,27).

The BIA variables measured were resistance (R), reactance (Xc), and PhA. R and Xc were considered as such or indexed to height (R/H and Xc/H) for bioelectrical impedance vector analysis (see below). R index was calculated as height^2/R (cm^2/ohm). The derived variables (TBW, FFM, and BCM) were estimated using the equations proposed by Kotler *et al.* (28). Fat mass (FM) was calculated as the difference between body weight and FFM.

Statistical Analyses

Data are reported as mean ± SD. One-way and two-way ANOVA were used to compare means. Bonferroni *post hoc* test was chosen for pairwise comparisons. Multiple regression analysis was used to evaluate the relationships between BIA and other variables, considering age, weight, and CrCl as continuous variables and gender, diabetes, and diuretic treatment as categorical variables. As regards bioelectrical impedance vector analysis, the bivariate 95% confidence intervals for mean impedance vectors of various groups were calculated, considering the bivariate normal distribution of R/H and Xc/H (18). Mean impedance vectors then were plotted in the resistance reactance (RXc) mean graph as arrowhead line segments with the corresponding 95% confidence ellipses. Hoteling's T^2 test for unpaired data was used to identify significant differences between impedance vectors. A two-tailed $P < 0.05$ was considered statistically significant for all statistical analyses.

Results

Main Characteristics and Nutritional Markers of Patients

After the exclusion criteria were applied, 84 patients with CKD (49 men and 35 women) of 104 consecutive patients who were screened were enrolled in the study. Four patients were excluded because of clinically detectable edema. Underlying renal disease was glomerular disease (13%), interstitial nephritis (20%), polycystic kidney disease (7%), diabetes (28%), hypertension (20%), and other or unknown conditions (12%).

All patients had mild to severe CKD (32 in stage 3, 31 in stage 4, and 21 in stage 5). Fourteen male and nine female patients had diabetes; 15 of them were on insulin treatment, and two were treated by oral glycemia-lowering drugs. Seventy-six (90%) patients were taking antihypertensive medications (angiotensin-converting enzyme inhibitors/angiotensin II receptor blockers [64%], calcium channel blockers [50%], α blockers [14%]; β blockers [12%] and other [11%]), with a mean prescription of 1.7 ± 0.9 drugs per patient (range 1 to 4); 39 (46%) patients were receiving diuretic treatment (Table 1). Other medications were allopurinol (24% of patients) and vitamin D

Table 1. Clinical characteristics and selected parameters of nutritional status in patients with CKD[a]

	Male	Female
n	49	35
Diabetes (%)	28	26
Use of diuretics (%)	49	43
Mean BP (mmHg)	112 ± 13	110 ± 14
Protein intake (g/kg per d)	0.79 ± 0.20	0.82 ± 0.23
Creatinine (mg/dl)	3.2 ± 1.8	2.8 ± 1.4
Albumin (g/dl)	3.8 ± 0.4	3.8 ± 0.3
Cholesterol (mg/dl)	198 ± 52[b]	234 ± 51
Triglycerides (mg/dl)	161 ± 74	181 ± 80
Transferrin (mg/dl)	223 ± 49	239 ± 50
Hemoglobin (g/dl)	12.5 ± 2.0	11.7 ± 1.7
CrCl (ml/min per 1.73 m^2)	27.8 ± 13.8	27.4 ± 13.0
Proteinuria (g/d)	1.6 ± 2.5	1.7 ± 2.4

[a]CKD, chronic kidney disease; CrCl, creatinine clearance.
[b]$P < 0.05$ *versus* female patients.

Table 2. General characteristics of patients with CKD and control subjects[a]

	Male		Female	
	Patients	Controls	Patients	Controls
n	49	298	35	306
Age (yr)	61.9 ± 11.7[b]	62.4 ± 11.2[b]	65.3 ± 13.9	65.2 ± 16.0
Height (cm)	166 ± 7[b]	167 ± 7[b]	153 ± 8	153 ± 7
Weight (kg)	72.1 ± 11.4[b]	72.1 ± 7.8[b]	61.1 ± 12.4	60.4 ± 7.9
BMI (kg/m^2)	26.1 ± 3.7	26.0 ± 2.3	26.1 ± 5.2	25.9 ± 3.4

[a]BMI, body mass index.
[b]$P < 0.05$ *versus* female patients.

Table 3. BIA variables and derived estimates of body composition in patients with CKD and control subjects[a]

	Male		Female	
	Patients	Controls	Patients	Controls
R (ohm)	465 ± 81[b,c]	492 ± 64[b]	550 ± 80[d]	574 ± 63
Xc (ohm)	39 ± 9[b,c]	53 ± 9[b]	43 ± 11[c]	58 ± 9
R index (cm^2/ohm)	61.5 ± 12.9[b,c]	57.5 ± 9.2[b]	43.5 ± 7.9[e]	41.4 ± 6.3
TBW (L)	41.8 ± 7.3[b,d]	40.1 ± 5.1[b]	30.0 ± 4.7[e]	29.0 ± 3.4
PhA (degrees)	4.84 ± 0.97[b,c]	6.22 ± 0.93[b]	4.51 ± 0.98[d]	5.78 ± 0.80
FFM (kg)	57.3 ± 9.2[b,d]	55.3 ± 6.5[b]	41.9 ± 6.9	40.8 ± 4.3
BCM (kg)	26.0 ± 4.3[b,c]	27.9 ± 3.4[b]	15.3 ± 2.5[c]	16.4 ± 2.1
FM (%)	20.3 ± 7.1[b,c]	23.2 ± 5.1[b]	30.4 ± 7.3	32.0 ± 6.3

[a]BCM, body cell mass; BIA, bioelectrical impedance analysis; FFM, fat-free mass; FM, fat mass; PhA, phase angle; R, resistance; TBW, total body water; Xc, reactance
[b]$P < 0.05$ *versus* female patients.
[c]$P < 0.01$ *versus* controls.
[d]$P < 0.05$ *versus* controls.
[e]$P = 0.06$ *versus* controls.

and/or calcium, erythropoietin, and drugs for heart diseases (each <10% of patients). At the time of enrollment, none of the patients had been prescribed a protein-restricted diet.

The values of common nutritional markers (Table 1) fulfilled, on average, the K/DOQI criteria for adequate nutrition in predialysis CKD (15). Considering major parameters of nutritional status, 16 patients had serum albumin concentrations <3.5 g/dl, but none of them had BMI <20 kg/m^2. Similarly, none of the seven underweight patients (BMI between 18 and 20 kg/m^2) showed hypoalbuminemia. Moreover, protein intake was >0.6 g/kg per d in all of the patients with low BMI or hypoalbuminemia.

BIA

In both patients with CKD and control subjects, age, height, and weight were slightly different between genders, whereas the mean BMI was similar (Table 2). As compared with control subjects, patients with CKD showed overt abnormalities of measured BIA variables, such as lower R, Xc, and PhA (Table 3). Clear-cut differences also emerged in bioimpedance vector analysis; in fact, significantly ($P < 0.001$) shorter and downsloping mean impedance vectors were observed in both male and female patients with CKD (Figure 1). In addition, R index (height^2/R), which is considered to be a variable that is related directly to TBW, was significantly ($P < 0.01$) higher in patients with CKD than in control subjects (on average, +7.0% in men and +5.1% in women). In particular, 27% of men and 20% of women were above the 90th percentile derived in control subjects for R index after adjustment for gender, age, and weight (data not shown). As regards the BIA-derived data, the differences in TBW ($P < 0.01$) were similar to those observed for R index, with a higher mean value in patients with CKD than in control subjects (men +1.8 L; women +1.0 L; Table 3); on the contrary, patients with CKD exhibited a reduction ($P < 0.01$) in both BCM (men −6.7%; women −7.7%) and FM (men −12.9%; women −5.0%).

Factors Influencing BIA in CKD

The patients in the three CKD stages (Table 4) were comparable for gender, age, BMI, and prevalence of diabetes; had similar serum albumin levels; and did not show significant differences with respect to other nutritional markers or daily protein intake. A lower creatinine excretion was in fact observed in CKD 5 compared with CKD 3 and 4 ($P < 0.05$). R, R index, and Xc were comparable among the three CKD stages. Consequently, TBW increased to a similar extent during CKD.

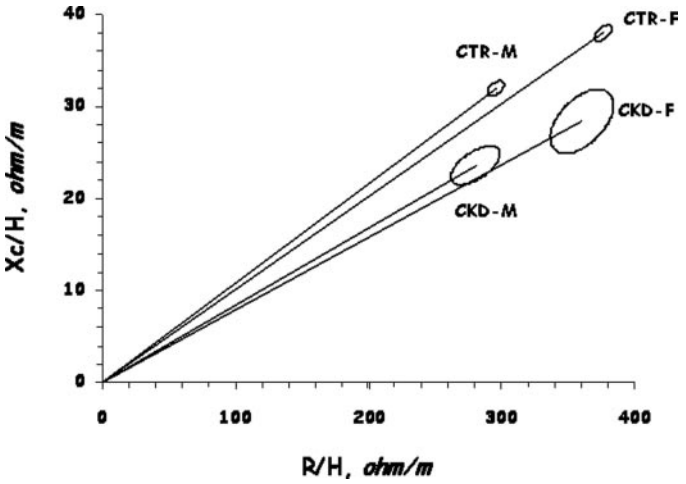

Figure 1. Bivariate resistance reactance (RXc) distribution (95% confidence interval) of bioelectrical impedance analysis (BIA) variables indexed to height (resistance, R/H; reactance, Xc/H) in patients with chronic kidney disease (CKD) and control subjects (CTR). M, male patients; F, female patients. $P < 0.001$ between CKD and CTR in both genders.

Indeed, no significant difference emerged in the mean impedance vector among the CKD groups. Conversely, compared with control subjects, PhA was reduced ($P < 0.01$) in each of the CKD stages (CKD 3 = 4.92 ± 1.01, CKD 4 = 4.71 ± 1.02, and CKD 5 = 4.50 ± 0.91 degrees).

The percentage of women was similar in CKD groups both with (nine of 23) and without (26 of 61) diabetes. Patients with diabetes were on average heavier (69.4 ± 11.1 *versus* 66.8 ± 13.6 kg; BMI 27.5 ± 4.3 *versus* 25.6 ± 4.3 kg/m^2) and exhibited a higher measured CrCl (30.5 ± 14.3 *versus* 26.5 ± 13.0 ml/min per 1.73 m^2). On the contrary, daily creatinine excretion was lower in patients with diabetes (13.1 ± 3.6 *versus* 14.9 ± 3.2 mg/kg per d; $P < 0.05$), whereas other markers of nutritional status, such as serum albumin, cholesterol, triglycerides, transferrin, and protein intake, were similar between the two CKD groups.

Patients with CKD and diabetes had lower values ($P < 0.01$) of R (468 ± 89 *versus* 512 ± 89 ohm), Xc (34.8 ± 8.1 *versus* 43.2 ± 10.4 ohm), and PhA (4.28 ± 0.81 *versus* 4.87 ± 1.00 degrees) than patients who had CKD without diabetes. In addition, the RXc mean graph (Figure 2) shows that the mean impedance vector of the CKD group with diabetes was shorter and more downsloping than in the CKD group without diabetes, which in turn was different from that of the control group.

To assess the simultaneous effects of various variables on BIA, we performed a multiple regression analysis that considered R or PhA as dependent variable and age, gender, weight, CrCl, diabetes, and diuretic treatment as potential predictors. The model demonstrated that predictors of R were age, weight, and diabetes. On the contrary, in addition to age and weight, CrCl, diabetes, and diuretic treatment all were significant predictors of PhA (Table 5).

Finally, when the other nutritional and laboratory variables were considered, no association was found in the whole CKD group between measured or derived BIA variables and main nutritional parameters, such as serum albumin, transferrin, cholesterol, triglycerides, and protein intake (data not shown).

Discussion

Although nutrition represents an important aspect in the treatment of renal patients, nutritional status has been evaluated scantily in predialysis CKD. Previous studies, which generated contrasting results, mostly were performed in advanced renal disease (8,11,12), often in small groups of patients, and,

Table 4. Individual and biochemical characteristics of patients in various CKD stages[a]

	CKD 3	CKD 4	CKD 5
n (%)	32	31	21
Gender (% female)	44	39	43
Age (yr)	65.8 ± 10.9	62.9 ± 11.6	60.1 ± 16.2
BMI (kg/m^2)	26.6 ± 4.4	26.1 ± 4.2	25.5 ± 5.3
BUN (mg/dl)	31.2 ± 10.3	47.0 ± 11.9[b]	56.8 ± 22.1[b,c]
Creatinine (mg/dl)	1.7 ± 0.3	2.9 ± 0.8[b]	5.2 ± 1.5[b,c]
Albumin (g/dl)	3.88 ± 0.28	3.76 ± 0.36	3.73 ± 0.56
Cholesterol (mg/dl)	230 ± 44	205 ± 53	199 ± 65
Triglycerides (mg/dl)	163 ± 75	175 ± 69	167 ± 90
Transferrin (mg/dl)	239 ± 41	238 ± 52	213 ± 53
Hemoglobin (g/dl)	13.1 ± 1.5	12.5 ± 1.6	10.2 ± 1.5[b,c]
CrCl (ml/min per 1.73 m^2)	42.2 ± 7.3	23.3 ± 3.5	11.8 ± 2.9[b,c]
Proteinuria (g/d)	0.9 ± 1.6	1.5 ± 2.1	2.3 ± 3.4[b]
Creatinine excretion (mg/kg per d)	15.1 ± 3.4	14.5 ± 3.6	13.1 ± 2.9[b]
Protein intake (g/kg per d)	0.88 ± 0.19	0.79 ± 0.19	0.79 ± 0.28

[a]BUN, blood urea nitrogen.
[b]$P < 0.05$ *versus* CKD 3.
[c]$P < 0.05$ *versus* CKD 4.

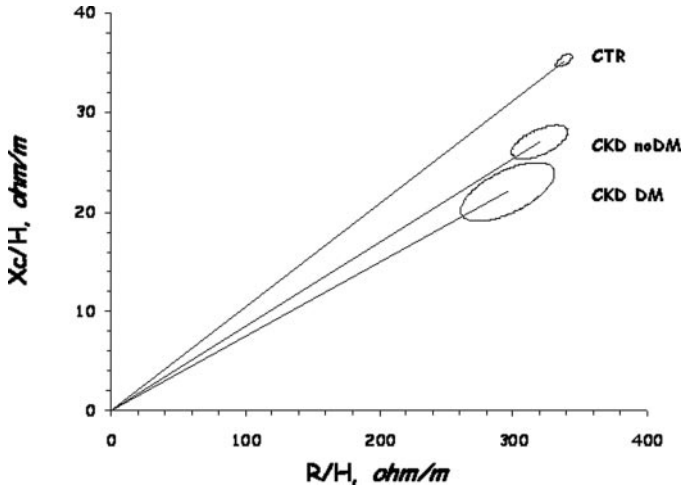

Figure 2. Bivariate RXc distribution (95% confidence interval) of BIA variables indexed to height (resistance, R/H; reactance, Xc/H) in patients (CKD) with diabetes (DM) and without diabetes (noDM) and CTR. $P < 0.001$ between CKD and CTR in both DM and noDM; $P < 0.001$ between CKD-DM and CKD-noDM.

Table 5. Multiple linear regression analysis with PhA as dependent variable in 84 patients who had CKD and were at the first referral to a nephrologist[a]

	β Coefficient	P
Age	−0.48	0.001
Weight	0.20	0.028
CrCl	0.29	0.008
Diabetes	−0.22	0.012
Use of diuretics	−0.20	0.024
Constant (5.770)		0.001

[a]For the entire model, $R^2 = 0.481$; $P = 0.001$. Gender and BMI did not enter the model.

generally, without including a control group (8,13). Furthermore, different methods were used, such as selected laboratory tests (5–13), evaluation of hand-grip strength (29), subjective global assessment (7,8), anthropometry (12), and dual-energy x-ray absorptiometry (10).

Previous studies have demonstrated that renal failure is a strong and independent risk factor for malnutrition, especially for GFR <30 ml/min per 1.73 m^2 (6,9). The impact of renal failure on body composition in our patients may be discussed taking into account that protein intake and nutritional status on average seemed adequate. Indeed, when the two major markers of malnutrition—body weight and serum albumin—were considered, only a small percentage of patients exhibited hypoalbuminemia or were underweight (BMI <20 kg/m^2), and none of them showed the composite condition of hypoalbuminemia-underweight. Therefore, these data suggest a low prevalence of overt abnormalities of nutritional status.

With regard to the assessment of body composition in CKD, BIA has attracted the interest of nephrologists, because it is a simple bedside method that provides information on both hydration status and body composition. In particular, in predialysis patients, it has been used to identify a threshold for apparent edema (21) and compared with deuterium oxide dilution and dual-energy x-ray absorptiometry to obtain adequate estimates of TBW and FFM, respectively (10,30); on the contrary, in only a few studies has it been used to evaluate specifically nutritional status (11–13).

Our controlled study included unselected patients who had mild to severe CKD and were at their first referral to a nephrologist. Under these conditions, overt abnormalities of the body bioelectrical properties clearly emerged. These abnormalities can be interpreted on the basis of the experimental evidence collected on BIA in various diseases (16,17,28). The presence of overhydration in patients with CKD was strongly suggested by an increased R index (as a result of reduced R), which is related directly to TBW (Table 3). Abnormalities of Xc and PhA also were apparent. Although the interpretation of PhA values still is ill-defined, it is commonly thought that PhA reflects the distribution between intracellular water and TBW (17,31). A decrease in PhA has been observed in male but not female patients without diabetes and with stage 5 CKD (12); in contrast, no statistical difference was found by others (11). Similarly, no significant changes in PhA were detected during a 9-mo follow-up in patients who had CKD and were on conservative therapy (13). Our study expands these observations, indicating that, in comparison with a control group, both male and female patients with CKD exhibit a marked decrease in PhA (−22%), with this difference persisting after adjustment for R index or TBW (data not shown). Some discrepancies with respect to previous results may be explained by the clinical characteristics of our patients (at first referral to a nephrologist, with no previous dietary treatment) or by the absence of matched control subjects in other studies. Indeed, patients with the same PhA may display a different hydration status. The RXc mean graph provides further interesting and more complete information on this issue. As already pointed out (32,33) and in agreement with previous data on peritoneal dialysis patients (34), the occurrence in patients with CKD of short mean impedance vectors that are associated with a small PhA strongly indicates the presence of fluid overload. Similarly, when conventional predictive BIA equations were used to predict body compartments, TBW was slightly increased in both male and female patients with CKD. Therefore, our study suggests that overhydration occurs in patients with mild to moderate CKD, even in the absence of clinically detectable edema. This is in agreement with a recent paper regarding the follow-up of patients with CKD (13) but not with the results of other reports (11,12). Again, it should be kept in mind that this study evaluated patients who were at the first referral to a nephrologist, whereas other studies included patients who were under regular nephrology follow-up (11,12).

Information on body compartments also can be obtained using conventional BIA equations. These derived measures, although not validated in CKD, are expected to be reasonably valid in patients without clinically detectable edema. Using

these estimates, BCM was lower in patients with CKD than in control subjects. A slightly lower FM also was present; this finding confirms the results of a previous paper (13). Furthermore, the decline in creatinine excretion observed in patients with stage 5 CKD and patients with diabetes indirectly suggests a decrease of skeletal muscle mass (6,35).

It is interesting that the results of our study indicate that changes in the bioelectrical characteristics of the body can be detected in an early phase of CKD. Indeed, in stage 3 CKD, PhA was much lower than that in control subjects (5.15 ± 1.06 and 4.61 ± 0.88 degrees in men and women, respectively); also the mean impedance vector was significantly shorter and more downsloping. Hence, an increased ratio between extracellular and intracellular water, coupled with overhydration, seems to occur when residual renal function still is relatively high. This finding is in agreement with a previous study that was conducted in patients with moderate to severe CKD (36). Diabetes also was associated with an increase in extracellular water, as indicated by the fact that patients with CKD and diabetes had lower PhA and a significantly different mean impedance vector with respect to patients without diabetes.

Finally, multiple regression analysis demonstrated that, after adjustment for other variables, PhA was narrower in the patients who were taking diuretics, perhaps because treatment had been started in patients who had edema and no complete correction of the increase in extracellular water had been achieved. However, it should be noted that PhA remained lower in the patients who did not have diabetes or receive diuretic therapy (5.28 ± 1.00 degrees in 18 men, 4.64 ± 1.18 degrees in 16 women) than in control subjects. This is an interesting finding because reduced PhA has been found to be associated with a low subjective global assessment score (13), which somehow may reflect malnutrition (17).

Conclusion

This study provides evidence that complex changes in body composition occur in patients who have CKD and are receiving conservative treatment. At first referral to an outpatient nephrology clinic, nonedematous patients with CKD show relevant alterations in the electrical properties of the body, such as a reduction of R, Xc, and PhA. The higher R index and the results of impedance vector analysis (RXc mean graph) strongly suggest the presence of overhydration. In addition, when considering the derived estimates of body composition, TBW tends to be higher, whereas BCM tends to be lower than that in control subjects. This impairment of body composition becomes evident from the early CKD stages, it is more marked in the presence of diabetes, and it occurs in the absence of overt signs of malnutrition. K/DOQI guidelines have underlined the need for sensitive measures of nutritional status to prevent malnutrition (7); this study suggests that BIA represents an attractive clinical tool to detect the early changes in body composition in patients with CKD.

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