Correlations of lean body mass with nutritional indicators and mortality in patients on peritoneal dialysis

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Detection of malnutrition in dialysis patients is important since this is a predictor of morbidity and mortality. Lean body mass (LBM) reflects the somatic protein store and this was measured by creatinine kinetics, anthropometry, and biometric impedance in 210 incident Chinese patients on continuous ambulatory peritoneal dialysis. The study was started in the third month of dialysis and the patients were followed for an average of 29 months. We devised three models of survival by combining the three different LBM measures with several nutritional markers and recognized outcome predictors. Follow-up was censored for transplantation or transfer to hemodialysis with an end point of death while on peritoneal dialysis. Statistical correlations were observed among the LBM values determined by all the three methods and these correlated significantly with both left and right hand grip strength but not with nutritional markers. LBM by creatinine kinetics, mean arterial pressure, and the calcium–phosphorus product were significant, independent predictors of death in one survival model. Anthropometry and bioelectric impedance were not significant predictors of death in the other two models. Our study suggests that LBM measured by creatinine kinetics, anthropometry, and bioelectrical impedance correlates well with the somatic protein store but not with the general nutritional status.

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It is well known that the early detection and diagnosis of malnutrition is critical in continuous ambulatory peritoneal dialysis (CAPD) patients since malnutrition is associated with high morbidity and mortality.[1,2] Lean body mass (LBM), which reflects the somatic protein store, is commonly used as a nutritional index for CAPD patients. The gold standard of LBM is the total body water (TBW) multiplied by 0.73, where the total body water is equal to the antipyrine distribution volume measured by using the tracer dilution method.[3] However, this method is laborious, invasive, and unsuitable for routine patient care. Therefore, noninvasive and indirect methods for measuring LBM, such as creatinine kinetics (LBM-CK), anthropometry (LBM-A), bioelectrical impedance (LBM-BEI), and dual-energy X-ray absorptiometry are increasingly applied for CAPD patients.

Although some studies have reported LBM measurement by simultaneously using two or three of the above-mentioned methods to evaluate the nutritional status of peritoneal dialysis (PD) patients,[4–21] few studies have considered whether LBM measured using different methods is related to other nutritional indicators. Keshaviah *et al.*[6] observed that LBM measured by CK correlated with the serum albumin (Alb), Scr, and nPCR levels, and they considered LBM-CK as a simple and convenient general nutritional index. However, Szeto *et al.*[7] reported that LBM-A and LBM-CK were poorly correlated with other nutritional indices, and they highlighted the importance of using multiple markers for assessing the various aspects of a patient's nutritional status. Further, Heimburger *et al.*[8] also did not observe good correlations of LBM-AM and LBM-CK with serum Alb. Thus, whether LBM measured by different methods is a general nutritional index remains unclear.

Conversely, LBM has not been adequately studied as a potential predictor of death, specifically among CAPD patients. Many logistic regression survival models involving PD patients did not include LBM in the analysis. To our knowledge, Heimburger *et al.*,[8] Trivedi *et al.*,[9] Szeto *et al.*,[10] and Chung *et al.*[11] used LBM-CK as a variable in survival analysis; however, not all authors demonstrated that LBM-CK is a strong negative predictor of death in CAPD patients. Further, whether LBM-A and LBM-BEI can predict

the outcome of CAPD patients has not been determined to date.

To our knowledge, the above-mentioned analyses regarding the correlations of LBM with other nutritional indicators and with the outcome of PD were not adjusted by sex, inflammation, and volume overload. However, the predictive power of various nutritional markers differed considerably between male and female patients.[12] Inflammation and volume overload are widely accepted as factors influencing the measurement of nutritional indices, particularly the visceral protein store, in PD patients.[22–24] Further, it is well known that volume overload can influence the LBM-BEI and LBM-A measurements.[13,14] In this study, we aim to identify whether LBM-CK, LBM-A, and LBM-BEI can reflect the general nutritional status adjusted by sex, inflammation, and volume status. Further, we aim to determine whether these measurements can predict the outcome of PD while considering certain recognized factors such as age, diabetes mellitus (DM), cardiovascular disease (CVD), inflammation, and volume status by performing retrospective and survival analyses on a large population of Chinese CAPD patients.

RESULTS

The patient demographics, etiology of renal disease, clinical characteristics, and biochemical parameters are summarized in Table 1. The mean duration of follow-up on PD was 29 ± 12 months. The outcomes were as follows: 30 of 210 patients died; four were transferred to hemodialysis; 11 had to undergo renal transplants; and 165 continued PD.

Subjective global assessment (SGA) on enrollment, that is, in the third month of PD, revealed that 36 patients (17.14%) were malnourished. The values (%) of LBM-CK, LBM-A, and LBM-BEI in the patients are shown in Table 2.

Our CAPD patients were divided into two groups depending on whether their serum C-reactive protein (CRP) levels were above or below $3\,mg\,l^{-1}$. Patients with $CRP \geqslant 3\,mg\,l^{-1}$ exhibited significantly lower dietery energy intake, dietery protein intake (DPI), and prealbumin (PA) values and a higher incidence of malnutrition diagnosed by SGA ($P<0.05$; Table 3). Partial correlation analysis revealed that the LBM-CK, LBM-A, and LBM-BEI values all correlated significantly with the hand grip strength (HGS) (R and L; $P=0.000$ for both) but not with the Alb, PA, transferrin, DPI and dietery energy intake, and SGA ($P>0.05$) adjusted by sex, CRP, and ECW/TBW (ECW, extracellular water). LBM-CK, LBM-A, and LBM-BEI correlated each other significantly, with r values ranging from 0.72 to 0.89 ($P=0.000$). All correlations are shown in Table 4.

Table 5 depicts the baseline and 'enrollment' variables that were observed to be independently associated with death in the three models ($P<0.05$). The stepwise procedure revealed that LBM-CK ($P=0.035$) and mean arterial pressure (MAP) ($P=0.027$) were negatively associated, while cCaP (product of corrected calcium and phosphate) ($P=0.016$) was positively associated with the risk of death in model 1. In models 2 and 3, cCaP ($P=0.022$ and $P=0.024$ respectively) and MAP ($P=0.004$ for both models) were both significantly associated with the risk of death. Table 6 lists all the variables that were excluded for the three models, of which LBM-A and LBM-BEI were not significant predictors of death in models 2 and 3, respectively.

Table 1 | Demographics, etiology of end-stage renal disease, clinical characteristics, and biochemical parameters of CAPD patients (n=210)

Age (years)	60.3 ± 14.1
Gender (male/female)	86:124
BMI ($kg\,m^{-2}$)[a]	23.52 ± 3.76
DM (n (%))	71 (33.8)
CVD (n (%))	64 (30.5)
Primary renal disease	
DM	58
Hypertension	56
Glomerulonephritis	58
Tubulointerstitial nephritis	27
Lupus nephritis	2
Other	19
RRF ($ml\,min^{-1}$)	2.00 (0–13.55)
Urine volume (ml)	640 (0–2700)
Ultrafiltration (ml)	400 (−200 to 3000)
TK_t/V	1.82 ± 0.54
Tccr(l per week per $1.73\,m^2$)	70.66 ± 29.41
Creatinine ($\mu mol\,l^{-1}$)	684.79 ± 251.74
TG ($mmol\,l^{-1}$)	2.21 ± 1.63 (0.32–12.1)
TCHO ($mmol\,l^{-1}$)	5.15 ± 1.41
Corrected calcium ($mmol\,l^{-1}$)	2.71 ± 0.50
Phosphorus ($mmol\,l^{-1}$)	1.59 ± 0.45
Product of corrected calcium and phosphorus ($mg^2\,l^{-2}$)	51.42 ± 15.93
iPTH ($pg\,ml^{-1}$)	145.5 (10–2000)
CRP ($mg\,l^{-1}$)	2.1 (0.17–94.73)
SBP (mm Hg)	133.73 ± 18.71
DBP (mm Hg)	78.16 ± 14.13
MAP (mm Hg)	97.11 ± 17.43

BMI, body mass index; CRP, C-reactive protein; CVD, cardiovascular disease; DBP, diastolic blood pressure; DM, diabetes mellitus; iPTH, intact parathyroid hormone; MAP, mean arterial pressure.RRF, residual renal function; SBP, systolic blood pressure; Tccr, total creatine clearance rate; TCHO, total cholesterol; TG, triglyceride; TK_t/V, weekly urea clearance.
[a]BMI was calculated as body weight (kg)/height (m^2).

Table 2 | Values of LBM-CK (%LBM-CK), LBM-BEI (%LBM-BEI), and LBM-A (%LBM-A)

Variables	Male (n=86)	Female (n=124)	Total (n=210)
$LBM\text{-}CK_{(1\,kg)}$	41.28 ± 9.13	30.86 ± 6.39	35.02 ± 9.14
%LBM-CK	66.13 ± 13.25	61.45 ± 13.08	63.54 ± 14.21
$LBM\text{-}BEI_{(1\,kg)}$	46.02 ± 9.04	33.65 ± 6.43	38.78 ± 9.54
%LBM-BEI	74.14 ± 9.24	67.23 ± 12.10	70.00 ± 12.01
$LBM\text{-}A_{(1\,kg)}$	54.90 ± 6.19	40.79 ± 6.29	47.50 ± 7.16
%LBM-A	87.33 ± 9.12	80.18 ± 11.12	83.40 ± 11.33

LBM-CK, lean body mass–creatinine kinetics; LBM-BEI, lean body mass-bioelectrical impedance; LBM-A, lean body mass-anthropometry; %LBM-CK, %LBM-A, and %LBM-BEI are LBM-CK, LBM-A, and LBM-BEI normalized to ideal body weight, respectively. Results are shown as mean ± s.e.m.

DISCUSSION

LBM, a marker of the somatic protein store, is a critical parameter for evaluating the nutritional status of PD

Table 3 | General nutrition parameters of CAPD patients with CRP $\geqslant$ 3 mg l^{-1} and CRP $<$ 3 mg l^{-1}

Variables	CRP $\geqslant$ 3l mg l^{-1} (n=74)	CRP $<$ 3l mg l^{-1} (n=136)	P (t, z or χ)
DEI (kcal per kg per day)	26.78 ± 6.65	29.27 ± 6.62*	0.015
DPI (g per kg per day)	0.78 ± 0.28	0.84 ± 0.23*	0.034
Alb (g l^{-1})	36.11 ± 4.63	37.25 ± 4.37	0.080
PA (mg per 100 ml)	213.00 (11.00–490.00)	311.00 (61.00–541.00)*	0
TF (mg per 100 ml)	346.25 (49.40–1239.20)	284.00 (9.06–1337.20)	0.372
HGS-R (n)	195.18 ± 89.05	209.07 ± 93.19	0.327
HGS-L (n)	211.03 ± 99.86	224.59 ± 96.11	0.373
SGA 2 or 3	18 (24.32%)	18 (13.23%)*	0.037

Alb, albumin; DEI, dietary energy intake; DPI, dietary protein intake; HGS-L, hand grip strength-left; HGS-R, hand grip strength-right; PA, prealbumin; SGA, subjective global assessment; TF, transferin.
Results are shown as mean ± s.e.m.; *P < 0.05 compared to CRP $\geqslant$ 3 mg l^{-1} group.

Table 4 | The relation of LBM measured by the three methods and general nutrition indices adjusted by sex, CRP, and ECW/TBW (n=210)

	LBM-CK	LBM-BEI	LBM-A
SGA			
r	−0.17	−0.17	−0.19
P	−0.08	−0.08	−0.07
Alb			
r	−0.06	−0.05	−0.04
P	−0.55	−0.61	−0.69
PA			
r	0.08	0.08	−0.01
P	−0.41	−0.43	−0.99
TF			
r	0.10	0.10	0.08
P	−0.34	−0.32	−0.42
DPI			
r	−0.12	−0.08	−0.13
P	−0.25	−0.44	−0.21
DEI			
r	−0.13	−0.12	−0.09
P	−0.21	−0.19	−0.34
HGS(R)			
r	0.53#	0.68#	0.62#
P	0	0	0
HGS(L)			
r	0.56#	0.69#	0.64#
P	0	0	0
LBM-CK			
r	—	0.76#	0.72#
P		0	0
LBM-BEI			
r	0.76#	—	0.89#
P	0		0
LBM-A			
r	0.72#	0.89#	—
P	0	0	

Alb, albumin; DEI, dietary energy intake; DPI, dietary protein intake; HGS(L), hand grip strength-left; HGS(R), hand grip strength-right; LBM-A, lean body mass-anthropometry; LBM-BEI, lean body mass-bioelectrical impedance; LBM-CK, lean body mass- creatinine kinetics; PA, prealbumin; SGA, subjective global assessment; TF, transferin.
#P < 0.001, *P < 0.05 between two indices.

Table 5 | Variables at the 'enrollment' point of PD determined to be associated with all-cause mortality by the multiple Cox proportional regression analysis in three models (significance level 0.05 or less, death=1)

	β	Wald	P	RR	95% CI for RR
Variables in model 1					
LBM-CK	−0.100	4.450	0.035	0.905	0.825 ~ 0.993
MAP	−0.040	4.890	0.027	0.960	0.926 ~ 0.995
cCaP	0.036	5.860	0.016	1.037	1.007 ~ 1.067
Variables in model 2					
MAP	−0.054	8.080	0.004	0.947	0.913 ~ 0.981
cCaP	0.034	5.253	0.022	1.035	1.005 ~ 1.065
Variables in model 3					
MAP	−0.055	8.366	0.004	0.946	0.911 ~ 0.982
cCaP	0.034	5.105	0.024	1.035	1.005 ~ 1.065

cCap, the product of corrected calcium and phosphate; LBM-CK, lean body mass-creatinine kinetics; MAP, mean arterial pressure.

patients. Since the antipyrine distribution volume, which is the gold standard of LBM, cannot be widely used for monitoring the nutritional status, certain alternative methods for routine LBM measurement have been applied for dialysis patients.[15] LBM measured by CK, A, and BEI is often used in clinical practice.

However, whether LBM is a general nutritional index or only a somatic protein index has not been clarified in the few related reports available;[4–21] these studies selected 1–3 methods for measuring LBM but obtained controversial results with regard to the correlations between LBM and other nutritional indicators. To our knowledge, Pearson correlation analyses were used in the above-mentioned studies and this type of analysis is unsuitable for cases wherein disturbing factors for measuring nutritional indicators exist. The first of such disturbing factors is sex. Stenvinkel *et al.*[12] observed that low HGS was an excellent independent outcome predictor in males but not in females, and they concluded that sex must be considered in studies related to nutrition and nutritional interventions in end-stage renal disease patients. The second disturbing factor is inflammation. Some authors demonstrated that indices of the visceral protein store, such as serum Alb, PA, and

Table 6 | Insignificant variables at the 'enrollment' point of PD excluded in three models by the multiple Cox proportional regression analysis

Variables in model 1	β	P	Variables in model 2	β	P	Variables in model 3	β	P
Age	0.065	0.799	Age	0.428	0.513	Age	0.529	0.467
BMI	0.072	0.789	BMI	0.144	0.705	BMI	0.177	0.674
CVD	0.194	0.660	CVD	0.224	0.636	CVD	0.129	0.720
DM	0.020	0.888	DM	0.101	0.751	DM	0.054	0.816
RRF	0.186	0.666	RRF	0.074	0.785	RRF	0.070	0.791
K_t/V	0.001	0.977	K_t/V	0.014	0.905	K_t/V	0.017	0.898
Tccr	0.126	0.723	Tccr	0	0.998	Tccr	0	0.999
CRP	2.764	0.096	CRP	2.102	0.147	CRP	2.303	0.129
iPTH	2.169	0.141	iPTH	1.332	0.248	iPTH	1.485	0.223
ECW/TBW	0.218	0.641	ECW/TBW	1.188	0.276	ECW/TBW	1.261	0.261
nECW	0.128	0.712	nECW	1.345	0.213	nECW	1.178	0.287
			LBM-A	0.185	0.667	LBM-BIA	1.685	0.194

BMI, body mass index; CVD, cardiovascular disease; CRP, C-reactive protein; DM, diabetes mellitus; ECW/TBW, extracellular water/total body water; iPTH , intact parathyroid hormone; K_t/V, weekly urea clearance; LBM-A, lean body mass-anthropometry; LBM-BEI, lean body mass-bioelectrical impedance; nECW, normalized extracellular water; RRF, residual renal function; Tccr, total creatine clearance rate.

transferrin, were negative reactant protein during the process of inflammation;[23,24] this result was confirmed in our study as shown in Table 3. These indices are now considered markers of inflammation. Thus, inflammation reflected by the serum CRP levels must be adjusted in correlation analyses in such studies. The third disturbing factor is the fluid status. Volume overload is known to lead to hypoalbuminemia in patients. In addition, the LBM-A and LBM-BEI measurements are possibly influenced by volume overload.[13,14] High ECW/TBW value has been confirmed to reflect fluid overload and to be associated with decreased technique survival;[16] therefore, we used this parameter as an indicator of the volume status and adjusted it in our study.

Our results revealed that LBM measured by CK, A, and BEI was not related to the visceral protein indices and to the protein and energy intake, and there was a minor but insignificant correlation between LBM and SGA. Only HGS, which is a good marker of the somatic protein store, exhibited a strong correlation with LBM measured by the three methods; this was similar to the results of the study by Heimburger and Wang *et al.*[8,17] The reason for no correlation between LBM-CK and DPI should be investigated. Theoretically, dietary proteins exert an important influence on creatinine excretion and consequently on LBM-CK measurement.[18,19,25] It has been hypothesized that different residual renal function (RRF) and dialysis doses lead to different levels of creatinine excretion despite similar protein intake levels, thus resulting in different LBM-CK values.

Another aim of our study was to clarify whether LBM measured by the three methods can predict the outcome of PD patients. A nutritional index is considered to be valuable if it can predict death in PD patients. For example, in previous studies, serum Alb, PA, and creatinine levels and SGA were confirmed to be predictors of death in PD patients; therefore, they are considered to be significant and valuable nutritional markers.[1,2] As stated in the introduction, only a few studies have included LBM as a parameter in logistic regression survival models involving dialysis patients.[9–11] To date, this is the fifth study to observe the prognostic role of baseline LBM-CK values in the outcome of PD patients. However, there are three points of difference between our study and previous studies. First, we combined many recognized prognostic factors with LBM in survival models involving a large PD patient population. Second, only a single nutritional index was considered in each model in order to easily determine its predictive effect. Third, based on previous literature, whether LBM-A and LBM-BEI have similar roles in predicting death is unclear.

Our data demonstrated that decreased LBM-CK is a significant predictor of death, while LBM-A and LBM-BEI are not predictors of death following adjustment by age, sex, body mass index (BMI), DM, CVD, RRF, K_t/V (weekly urea clearance), total creatinine clearance (Tccr), CRP, cCaP, intact parathyroid hormone (iPTH), ECW/TBW, normalized ECW (nECW), and MAP. The possible reasons for this are as follows. First, LBM-CK is a marker of the somatic protein store, similar to the Scr and creatinine indices, which have been confirmed as strong prediction factors for PD patients in other reports.[2,26,27] Second, previous studies have suggested that LBM-CK measurement was unaffected by a patient's fluid status;[15] therefore, the Dialysis Outcome Quality Initiative guidelines recommend routine use of the CK method.[28] Third, Johansson[18] reported that as compared to measurement of the total body potassium, LBM-CK significantly underestimated LBM by 2–14 kg. Further, as compared to the antipyrine distribution volume, which is the gold standard for measuring LBM, LBM-CK underestimated LBM by 6.9 kg.[3] It should be considered that the correlation between LBM-CK and the outcome of CAPD patients may be related to the fact that this method, as mentioned earlier, underestimates the LBM and may falsely indicate patients as malnourished. Fourth, LBM-BEI is calculated based on the TBW measurement; thus, the LBM may be overestimated in a patient suffering from fluid overload.[13] This may explain why although the LBM measured by BEI was high, there was no significant difference in the patient outcome. Fifth, LBM-A,

which is an operator-dependent method, may not be sufficiently sensitive to predict death in dialysis patients because Nelson *et al.*[29] observed no visible difference in the triceps and subscapular skinfolds between age-, sex-, and race-matched healthy controls and CAPD patients. In addition, the equation by which LBM-A was calculated in our study was developed for patients from the UK and may not have been appropriate for Chinese patients.

The next point is why the baseline single values of LBM-CK can predict the outcome of PD patients. As reported by Johansson *et al.*,[18] LBM-CK values may be influenced by several factors, including dietary meat intake, physical activity, hormonal balance, diurnal glomerular filtration rate variations, and catabolic states; this would explain the unacceptable high coefficient of variation observed for LBM, that is, 14.2%. For this reason, repeated examinations should be conducted over several days in order to minimize the effects of 'timing errors,' and this reduces the convenience of this method for standard use. However, recently, Trivedi *et al.*[9] provided a good explanation as to why single values of LBM play an important role in the outcome; they reported that death could be predicted in PD patients based on both the baseline value and the weighted time average of LBM-CK This suggests that the baseline single values and the weighted time average values of LBM-CK are similar for most PD patients. In our study, the variation in the LBM-CK was relatively minor, probably because the follow-up time was only two and a half years. Therefore, serial monitoring should be recommended instead of a single LBM-CK measurement in each PD patient in order to reduce the systemic errors in LBM measurement occurring in most centers and thus better determine variations in the somatic protein store.[30,31]

Our study posed certain limitations. First, the study was based on observations, and the follow-up time was relatively short. Second, the role of LBM-CK in predicting survival was not evaluated using the gold standard of LBM. Furthermore, this study was conducted at a single center; therefore, the case-mix characteristics may not have been representative of the general PD population.

In conclusion, our results revealed that the LBM measured by CK, A, and BEI correlated well with the somatic protein store but not with other nutritional indicators of the visceral protein store (serum Alb, PA, and transferrin) and with the dietary nutrient intake. Further, LBM-CK but not LBM-A and LBM-BEI could predict death in PD patients. Therefore, LBM-CK can be considered as a more valuable method for examining LBM to estimate the outcome in PD patients.

MATERIALS AND METHODS

Patient selection and follow-up

This study comprised 210 patients in the third month of PD at the PD centers of Peking University First Hospital; the patients were enrolled from January 2002 to June 2005. Baseline demographic and clinical data were collected, including data of age, sex, BMI, etiology of end-stage renal disease, DM, and CVD. CVD included congestive heart failure, angina pectoris, old myocardial infarction, and cerebral infarction, as described in a previous study. The biochemical index, dialysis adequacy, and general nutrition status of the 210 patients were simultaneously examined during the third month of dialysis as the enrollment values. Brachial blood pressure was measured by using a standard method. Systolic and diastolic blood pressure were recorded, and the MAP was calculated by using a standard method. The patients were followed-up until November 2006. Follow-up was censored on transplantation or transfer to hemodialysis. The end point was death on PD. All the patients were dialyzed by using glucose lactate-buffered PD solutions (Baxter Health Care Inc., Guangzhou, China).

Laboratory data

Biochemical index. Biochemical indexes were analyzed using Hitachi chemistry analyzer, and include Alb, PA, transferrin , urea nitrogen (BUN), creatinine, triglyceride (TG), cholesterol (TCHO), calcium, and phosphate. cCaP was calculated. iPTH was determined by the immunoradiometricassay.

Dialysis adequacy. Twenty-four-hour dialysate and urine collection were performed to calculate the fluid removal and solute clearances. RRF was estimated using the average renal clearance of urea and creatinine. K_t/V and Tccr were calculated using standard methods. The distribution volume of urea (V), which is generally assumed to be equal to TBW, was calculated from the Watson equation.

Inflammation marker. Serum high-sensitive CRP was measured by immune rate nephelometric analysis. The detection limit of CRP was $0.06\,mg\,l^{-1}$. Serum CRP $\geqslant 3\,mg\,l^{-1}$ was seen to be abnormal.

Subjective global assessment. SGA was performed to evaluate the overall protein–energy nutritional status by experienced research staff blinded to all clinical and biochemical variables of the patients.[10] On the basis of evaluation, each patient was scored as 1 = normal, 2 = mild to moderate malnourished, and 3 = severe malnourished.

Protein and energy intake. A continuous 3-day dietary was recorded on a self-completed food diary. Then, DPI and dietery energy intake were calculated and normalized for actual body weight. The total calorie intake includes intake from dietary and dialysate.

Hand grip strength (HGS). HGS was evaluated in both the dominant and non-dominant arm using the dynamometer, which was repeated three times and the greatest value was recorded in Newton (N).

LBM. (1) LBM-CK: LBM was measured by the creatinine kinetics method as described below. Dialysate and urine collections for a 24-h period were obtained, and urea and creatinine in dialysate, urine, and serum were examined at the same time. LBM-CK was calculated according to the formula recommended by Blake:[32]

$$\mathrm{LBM-CK} = 7.38 + 3.29\times(\mathrm{CE} + \mathrm{CD})$$

where CE is creatinine excretion in millimoles per day and CD is creatinine degradation in millimoles per day. Creatinine excretion and degradation were calculated using the formulas

$$\mathrm{CE} = \mathrm{UCO} + \mathrm{DCO}$$
$$\mathrm{CD} = 0.04\times\mathrm{PC}\times\text{body weight}$$

where UCO is the urinary creatinine output in millimoles per day, DCO is the dialysate creatinine output in millimoles per day, and PC is the plasma creatinine in micromoles per liter.

(2) LBM-A: Anthropometric measurements were taken in millimeters by trained observers using standard skin-fold calipers. The observers were blinded from the results obtained by applying the creatinine kinetics formula. Measurements included biceps, triceps, subscapular, and supra-iliac skin-fold thickness. For each site, the observers obtained three readings, the average value of which was used for further calculations. Lean body mass (in kilograms) by the anthropometric method (LBM-A) was computed using standard formulas:[19]

$$\text{total skin fold} = \text{biceps} + \text{triceps} + \text{subscapular} + \text{suprailiac}$$

$$\text{body density (male)} = 1.161 - [0.0632 \times \log(\text{total skin fold})]$$

$$\text{body density (female)} = 1.1581 - [0.072 \times \log(\text{total skin fold})]$$

$$\%\,\text{body fat} = [(4.95/\text{body density}) - 4.5] \times 100$$

$$\text{LBM} - \text{A} = \text{body weight} \times (1 - \%\,\text{body fat}/100)$$

(3) LBM-BEI: Multiple-frequency bioelectrical impedance analysis was performed using the hydra analyzis (Xitron Technologies, San Diego, CA, USA). The procedure is described in detail elsewhere. Briefly, after a patient drained the dialysate and was in a supine position for at least 10 min, the standard tetrapolar electrodes were placed on the dorsum of the wrist and anterior aspect of the ankle on the left side of the body. Three consecutive measurements were performed during a 2-min period, with recording of values for ECW, intracellular water , and TBW. Height nECW was calculated. Resistance values obtained were then used to calculate LBM using a software package provided by the manufacturer.

LBM-CK, LBM-A, and LBM-BEI were all normalized to ideal body weight (IBW, IBW = height (cm) − 105) and subsequently represented as %LBM-CK, %LBM-A, and %LBM-BEI.

Statistical analyses

Statistical analysis was performed using the SPSS software, version 11.0 (SPSS Inc., Chicago, IL, USA). Continuous variables were expressed as the mean value ± s.d., whereas categorical variables were expressed as a percentage or ratio. Partial correlation analysis was used to analyze the relationship of the LBM measured by the three methods adjusted by sex, CRP, and ECW/TBW, respectively, with other nutritional parameters. Recognized outcome predictors combined with LBM-CK, LBM-A, or LBM-BEI were considered in Cox proportional regression (forward conditional) models to determine whether LBM measured by the three methods could predict the outcome. Thus, three Cox proportional regression models were constructed. In model 1, the variables included age, sex, BMI, DM, CVD, RRF, K_t/V, Tccr, CRP, cCaP, iPTH, ECW/TBW, nECW, MAP, and LBM-CK. In model 2, they included age, sex, BMI, DM, CVD, RRF, K_t/V, Tccr, CRP, cCaP, iPTH, ECW/TBW, nECW, MAP, and LBM-A. In model 3, they included age, sex, BMI, DM, CVD, RRF, K_t/V, Tccr, CRP, cCaP, iPTH, ECW/TBW, nECW, MAP, and LBM-BEI. The final models comprised those variables that remained in the model with a significance level of 0.05. $P < 0.05$ indicated statistical significance.

DISCLOSURE

There are no interests to be disclosed.

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